Congenital Defects in 18th and 19th Century Populations from Rural and Urban Northeast England

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CONGENITAL DEFECTS IN 18TH AND 19TH CENTURY POPULATIONS FROM RURAL AND URBAN NORTHEAST ENGLAND

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ABSTRACT

In England, the 18th and 19th centuries marked an increase in urban living and the development of industrialisation. The movement of large numbers of individuals into newly created urban, industrial centres led to a decline in the standard of living conditions. In overcrowded towns, infectious disease easily spread amongst the improperly fed masses exposed to air and water pollution from nearby factories. To investigate the effects of these poor living conditions on populations in the post-medieval period, the prevalence of congenital defects, anomalies present at or before birth, were chosen for study in skeletal remains. Using an analysis of the prevalence of congenital defects, the hypothesis tested was that there should be a greater prevalence of congenital defects in people in urban centres due to the inferred poor state of health present there at the time compared to individuals from rural areas who may not have been as heavily exposed to unsanitary environmental conditions.

This research focused on populations from four sites in Northeast England. The two urban sites were the Quaker burial ground, Coach Lane, North Shields (1711-1857 AD) and St Hilda's, Coronation Street, South Shields (1816-1856 AD), both in Tyne and Wear. The two rural sites were St Michael and St Lawrence, Fewston (post-medieval-1896 AD) and St Martin, Wharram Percy (1540-1850 AD), both in North Yorkshire. Collected data showed that there was no statistical difference between prevalence rates at the urban and rural sites for individual or combined defects. This may indicate that the quality of the living conditions were similarly detrimental to health at both site types and raises the issue of how urban and rural can be better defined for the post-medieval period. Furthermore, these findings call into question the use of congenital defects as markers of overall health unless combined with "stress" indicator data and research into past living conditions.

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VOLUME I

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LIST OF ABBREVIATIONS

#	number
c	deciduous canine
C	permanent canine
CO	cribra orbitalia
DEH	dental enamel hypoplasia
DJD	degenerative joint disease
F	female
?F	potential female
Fe	Fewston
i1	first deciduous incisor
I1	first permanent incisor
i2	second deciduous incisor
I2	second permanent incisor
L	left
M	male
?M	potential male
m1	first deciduous molar
M1	first permanent molar
m2	second deciduous molar
M2	second permanent molar
M3	third permanent molar
MNI	minimum number of individuals
mos	months
NS	North Shields
obs	observable
PH	porotic hyperostosis

PM	post-mortem
PM1	first permanent premolar
PM2	second permanent premolar
PNBF	periosteal new bone formation
R	right
SS	South Shields
U	unable to determine sex
wiu	weeks in utero
WP	Wharram Percy
yrs	years

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CHAPTER 1
INTRODUCTION

'Abnormal they certainly are in the sense of not being the usual form in which a given species manifests itself, but they are not deformed.'
(Hawthorne Wilder, 1908:356)

1.1 Definitions and Research Background

1.1.1 Definitions

A congenital defect, commonly called a birth defect, is any physical formation present at or before birth that differs from what is deemed normal (Aufderheide and Rodríguez-Martín, 1998; Barnes, 1994, 2008, 2012a; Dellicour *et al.*, 2013; Groisman *et al.*, 2013; Office for National Statistics, 2010; Ortner, 2003; Spranger *et al.*, 1982; Turkel, 1989; WHO, 2012). In some instances, the defect may not be observable at birth but rather the aetiological factors are in place for it to develop in later life. The defect is therefore only observable after bone development or organ formation is completed. The terms defect and anomaly can both imply a deviation from the normal form and are frequently used interchangeably in clinical and bioarchaeological studies, therefore both will be used in this thesis.

Congenital defects can range from so mild that there are no symptoms in the affected individual to so severe that the anomalies are incompatible with life (Aufderheide and Rodríguez-Martín, 1998; Barnes, 2012b; Dellicour *et al.*, 2013; Groisman *et al.*, 2013; Ortner, 2003; Turkel, 1989; WHO, 2012). As a group, these defects are reasonably common but the majority of the individual anomalies are rare, both today and in the past (Barnes, 2008; Office for National Statistics, 2010; Turkel, 1989). Teratology, literally the study of monsters or marvels, is the study of congenital defects and was coined in the first half of the 19th century by Isidore

Geoffroy Saint-Hilaire, an early researcher of congenital defects (Blumberg, 2009; Lancaster, 2011). From the same root, a teratogen is any agent that causes a congenital defect (Lancaster, 2011).

Non-metric traits are minor developmental variants that are generally considered to be normal variations of the skeleton (Berry and Berry, 1967; Saunders and Rainey, 2008). Commonly, confusion has arisen as to where the line is drawn between congenital defects and non-metric traits as both are present from birth and can have similar causes (*e.g.* genetics or radiation) (*ibid.*). The difference is that non-metric traits are dichotomous because they can be scored as present or absent (Saunders and Rainey, 2008). Congenital defects are continuous in that they can have various levels of expression and there can be a gradation of changes. The confusion between non-metric traits and congenital defects has lead to misreporting of congenital defects as non-metric traits in bioarchaeological research. Furthermore, it may have ramifications for determining the aetiology of these traits/defects as well as for their usefulness in bioarchaeological research. As non-metric traits are largely attributed to genetic aetiologies or causes, environmental factors may be overlooked for causing any congenital defects assigned to that category. Similarly, for congenital defects attributed to the category of non-metric traits, differences in environment and living conditions would be excluded as potential causes for these defects in bioarchaeological research when comparing changes to populations over time. Therefore, conditions that are continuous need to be classified as congenital defects rather than as non-metric traits to be able to investigate both genetic and environmental causes of changes to the skeletons of past populations.

1.1.2 Background to Congenital Defects: Past and Present

A. Documentary Evidence

Congenital defects have been found worldwide throughout history and are still common today. Knowledge that congenital defects existed in the past can be gained from popular and scholarly written texts, focusing here on evidence from

post-medieval Britain. It is notable that the general public, along with surgeons and other researchers of this period, were exceptionally interested in congenital defects (Bates, 2005). Across Europe, the 16th and 17th centuries saw unusual births and individuals with congenital defects ("monsters") as signs or messages from God and, as such, had to be closely examined in an effort to "read the message" (Anderson, 2000; Asma, 2009; Bates, 2000, 2005). The term monster comes from the Latin *monstrum* meaning portent or warning (Bates, 2005; Tonelli, 2012) and these births were often thought of as harbingers of doom or some great calamity (Asma, 2009). They were also commonly thought to be physical punishments for some sin or moral misdeed of the parent(s) (Asma, 2009; Bates, 2000, 2005; Blumberg, 2009).

News of an individual being born with a congenital defect spread rapidly through the dissemination of broadsheet newspapers in Britain, made possible after the invention of the printing press allowed for quick and cheap printing (Bates, 2000, 2005; Blumberg, 2009). Although broadsheets were not the only written source for the public to learn of "monstrous" births in Britain as publications such as the *Gentleman's Magazine* reported on these unusual births as well (de Montluzin, 2002). The first broadside in England detailing a "monstrous" birth was printed in 1552. The text of broadsheets was commonly written in the form of a rhyming ballad, making it easier to read or interpret through song, an important consideration when much of the population was illiterate to semi-literate (Bates, 2000; Blumberg, 2009). Especially popular were broadsheets detailing the birth of conjoined twins (Figure 1.1) (Bates, 2005). Of the 3,081 broadsheets registered with the Stationers' Company in London starting in 1557, seventeen were about "monstrous" births (Bates, 2000, 2005). There were no broadsheets printed about illnesses, accidents, or other medical matters but also none printed about dwarfs, giants, or other "freaks" appearing in the sideshows of the time (*ibid.*). This disparity demonstrates that "monsters" were not popular due to their medical interest, rather they were seen as special, showing the power or wrath of God (Bates, 2000). They were defective in form, unlike pygmies or giants who were abnormal in stature but were otherwise considered to be normal humans.

Figure 1.1: A broadsheet depicting conjoined twins born in 1566 in Buckinghamshire (Bates, 2000).

The "announcements" of infants with congenital defects on broadsheets often served as an invitation for people to come and view the affected child (Bates, 2000). The public's moral view on the deformed child, that he or she was born that way due to some sin of the parent, did not stop them from willingly paying to view the child (Blumberg, 2009). This could serve as a welcome source of income for a poor family, and some adults with congenital defects exhibited themselves for their own financial gain (Bates, 2000, 2005; Blumberg, 2009; Wilson, 2002). There is no evidence that individuals with congenital defects on exhibition were ridiculed or feared, as the tendency was to emphasise the accomplishments of the "monster" (Bates, 2005). The exhibition of individuals with defects in the 16th and 17th centuries resulted in parents taking their affected children on the road to reach a wider audience, which eventually evolved into the sideshows of the 18th and 19th centuries (Asma, 2009; Bates, 2005; Blumberg, 2009).

Turning from the popular to the scholarly literature, Ambroise Paré composed an early essay on the existence of various congenital defects (as well as some outlandish non-existent monsters) and their causes in 1573. While most causes were attributed to spiritual beings (*i.e.* the will or wrath of God, devils, and demons), genetic and environmental factors (*i.e.* 'hereditary or accidental illness' (Paré, 1573:4) and restriction of the fœtus *in utero*) were also blamed. His definition

of hereditary disease was basically "like produces like," and while his reasoning was sound for some defects (*e.g.* individuals with dwarfism give birth to children with dwarfism) it was not in all cases (*e.g.* individuals with leprosy give birth to children with leprosy). Paré's work helped pave the way for a more scientific approach to the recording and interpretation of these defects.

The Sick Woman's Private Looking-Glass written by John Sadler in 1636 for women concerned about the prospect of giving birth to a "monster" stated that while some children were born with congenital defects due to the sins of the parents, many were also born due to "a trick of nature," something was wrong physically rather than spiritually (Asma, 2009). By the end of the 17th century, births of individuals with congenital defects were starting to be viewed more in a scientific light than in a religious one (Asma, 2009; Bates, 2005). There was more research into the natural rather than automatically attributing everything to God and his will. Research in the 18th and 19th centuries detailed a wide variety of defects and laid the groundwork for modern research on the subject (Asma, 2009; Blumberg, 2009). John Hunter working in the 18th century hypothesised that there was an internal code controlling the formation of humans or animals (Asma, 2009). It was when this code went astray that malformed humans and animals were born (an intrinsic or genetic factors model). He also realised that defects occurring in certain areas of the body only developed during the gestational period scheduled for the formation of that area (*ibid.*). Etienne Geoffroy Saint-Hilaire (the father of Isidore mentioned in Section 1.1.1), determined in the early 19th century that external forces such as trauma or extreme heat can lead to the formation of developmental defects (an environmental or extrinsic factors model) (*ibid.*).

B. Bioarchaeological Evidence

Another vital and primary source for the study of congenital defects in the past is skeletal remains from archaeological contexts. In the bioarchaeological literature, numerous case studies of individuals with evidence of congenital defects can be found (*e.g.* Anderson, 2003; Bašić *et al.*, 2012; Keenleyside, 2012a, b;

Lieverse *et al.*, 2012; Murphy, 1996; Pany and Teschler-Nicola, 2007; Pedersen and Antón, 1998; Usher and Christensen, 2000). While these studies are important for disseminating information on the presence of certain conditions at a certain point in time or in a given region, or for better defining osteological diagnoses, little more can be learned from these reports (Anderson, 2000; Mays, 2012a; Roberts and Manchester, 2005; Turkel, 1989; Wood *et al.*, 1992). Very few population studies have been undertaken to establish frequencies spatially or temporally (*i.e.* Barnes, 1994; Kase, 2010; Masnicová and Beňuš, 2003; Murphy, 2000; Sture, 2001). Population studies are important as they can show the frequency of congenital defects as a result of changes in environment, cultural practices (*e.g.* marriage practices, differential treatment of women, sanitation), genetic relationships, and immigration (Brothwell, 1963; Mays, 2012a; Turkel, 1989). This is especially true when combined with research into possible teratogenic agents (environmental factors that cause congenital defects) and contemporary archaeological and/or historical records.

C. Modern Day

In modern populations, notification systems have been set up to monitor the frequency of defects. The World Health Organization (WHO) reports a worldwide incidence of 1 in 33 infants being born with a congenital defect, resulting in 3.2 million defect-related disabilities per year (WHO, 2012). Congenital defects are most commonly found amongst lower socioeconomic families and countries, with 94% of "serious" birth defects occurring in lower and middle income countries (WHO, 2012). According to the WHO, expectant mothers in these lower and middle income countries are more likely to be exposed to malnutrition, infections, and pollutants (*e.g.* heavy metals and pesticides), while there is also a greater tendency for alcohol abuse.

The Office for National Statistics (UK) reports one or more congenital defects in 1.34% of live and stillbirths occurring in regions of England and Wales that reported to the National Congenital Anomaly System in 2008 (the most

recently available data) (Office for National Statistics, 2010). In the Northern notification region (which includes Northumberland, Durham, Tyne and Wear, and Carlisle) 514 live or stillbirths were reported as having one or more congenital defect. This accounted for a rate of 152.0 per 10,000 live or stillbirths, which was the highest recorded for any English region (although the statistics are not available on a regional basis for the whole country) (*ibid.*). Unfortunately the Office for National Statistics report does not explain any causation for the defects recorded or the high prevalence rate occurring in the North.

Using data from the Office of National Statistics (UK) from 2008 and data from the International Clearinghouse for Birth Defects Surveillance and Research (ICBDSR) from 2010, it is possible to compare data between the UK and two industrialising countries/regions, India and South America. The overall rate of infants born with at least one congenital defect was not available for India or South America, but data was available on two of the most common congenital defects to affect the skeleton. For spina bifida, the Office for National Statistics (UK) reported a prevalence rate of 1.3 per 10,000 births, compared to the higher rates of 10.96 and 8.60 per 10,000 births in India and South America respectively (ICBDSR, 2012; Office for National Statistics, 2010). For cleft palate, meanwhile, India reported the lowest rate of the three showing a rate of 1.85 per 10,000 births with rates of 4.5 per 10,000 births in the UK and 5.02 per 10,000 births in South America (*ibid.*).

As can be seen, the prevalence of congenital defects varies depending on location and socioeconomic status. Expectant mothers from urbanising and industrialising nations may have limited access to modern health care and be more greatly exposed to environmental and living conditions that are hazardous to health (WHO, 2012). This may make children in these nations more liable to be born with congenital defects. This is important because the conditions experienced in these developing nations are likely to be similar to those in 18th and 19th century Britain at the start of the Industrial Revolution (Dellicour *et al.*, 2013; McDade and Adair, 2001; McMichael, 1999; Oliveira *et al.*, 2011; Peters, 1999; WHO, 2012). Furthermore, given this link between poor living conditions and congenital defects,

individuals living during the start of the Industrial Revolution in urbanising and industrialising areas may show higher frequencies of these anomalies than those living in more rural regions where exposure to factors such as air pollution and infectious disease may have been lessened. This link between congenital defects and living conditions as well as the possibility of differential living conditions in urban versus rural areas are the focus of this thesis as will be discussed further in Sections 1.2 and 1.3.

1.2 Research Rationale

The impact of environmental teratogens on the development of congenital defects in the past is important as this is still a concern today. Teratogenic agents include infectious disease (*e.g.* measles, syphilis), malnutrition (*e.g.* lack of folic acid or an excess of Vitamin A), excessive alcohol consumption, drug use (prescription or recreational), air and water pollution, radiation (*e.g.* x-ray or nuclear) and chemicals (Abbott, 2010; Bánhidy *et al.*, 2011; Botting *et al.*, 1999; Connor and Ferguson-Smith, 1997; Czeizel *et al.*, 2008; Gianicolo *et al*, 2012; Gluckman and Hanson, 2005; He *et al.*, 2011; Schoenwolf *et al.*, 2009; Uriu-Adams and Keen, 2010). While some of these factors would have been rare or non-existent in the past (*i.e.* drug use, radiation, and certain chemicals), others such as polluted air and water, malnutrition, and certain infectious diseases, may have played a significant role in the development of congenital defects in both individuals and the wider population.

The 18th and 19th centuries (the period of this study) continued the trend started in the late medieval period of industrial development and an increased urban population (Briggs, 1990; Brimblecombe, 1978, 1987; Ellis, 2001a; Holt and Rosser, 1990; Roberts and Cox, 2003). High population densities, poor living conditions, and inadequate waste disposal systems in 18th and 19th century urban areas were often associated with high rates of infectious disease and unclean drinking water (Briggs, 1990; Daunton, 1983; Ellis, 2001a; Hardy, 1993; Manchester,

1992; Roberts and Cox, 2003; Waldron, 1989). Intensified coal use due to increased domestic and industrial demand in the post-medieval period (*c.*1550-1850 AD) would also have affected air quality and generated different types and amounts of particulates in the environment (Brimblecombe, 1978, 1987; Roberts and Cox, 2003; Thornborrow, 1988; Warren, 1980). Furthermore, malnutrition could have been prevalent in certain areas of towns due to poverty and the high cost of food (Moffat and Rosie, 2005; Roberts and Cox, 2003; Waldron, 1989). These poor living conditions have been linked to increased frequencies in congenital defects in the past. Sture (2001) examined urban and rural populations from the medieval period in England and found higher frequencies amongst the urban dwellers. Moving slightly later in time, Kase (2010) conducted a literature review of congenital defects found in individuals at late and post-medieval sites across Britain. Her study showed an increase in congenital defects when moving from the medieval to the post-medieval period. Additionally, there was a higher frequency of congenital defects amongst urban populations when compared to their rural counterparts.

Many of the compromised living conditions found during the 18th and 19th centuries in Britain can currently be found in industrialising nations around the world and are known to cause congenital defects (Dellicour *et al.*, 2013; McDade and Adair, 2001; McMichael, 1999; Oliveira *et al.*, 2011; Peters, 1999; WHO, 2012). In understanding the frequencies of congenital defects in the past, there are potential applications for combating the development of these defects in living populations. This may apply particularly to people living in developing countries, and could include education programmes aimed at young women to combat the spread of infectious disease and the creation of clinics specializing in maternal health and nutrition.

1.2.1 Post-Medieval Archaeology

A focus on the post-medieval period was chosen as this period has been largely neglected in archaeological research of any type in Britain (Cherryson *et al.*, 2012; Matthews, 1999; Petts and Gerrard, 2006; Roskams and Whyman, 2007). Frequently it seems to be assumed that any pertinent information regarding this

period can be found in existing historical records, implying it is not worth the expense and time to seek out archaeological data (Bayley and Crossley, 2004; Matthews, 1999). Archaeological evidence for this period is being missed as post-medieval buildings are being demolished before they can be recorded, while buried artefacts are often intentionally overlooked during excavations (Matthews, 1999; Petts and Gerrard, 2006). Additionally, skeletal remains from burial grounds are commonly cleared by a commercial cemetery-clearance company for reburial rather than being studied by bioarchaeologists (Cherryson *et al.*, 2012; Mays, 1999). Despite the beliefs in the unimportance of archaeological research into the post-medieval period, it can in fact offer much to the knowledge for the era. Archaeological artefacts and human remains can fill gaps in knowledge highlighted in historical documentation, identify some of the bias inherent in written records (not that archaeological research is without inherent bias), and lead to a fuller narrative of the past (Clark, 1999; Matthews, 1999; Mitchell, 2012; Shuler, 2011; Symonds, 2006). Furthermore, individuals such as the poor or rural dwellers and their possessions, often left out of the written record, can be found archaeologically.

1.2.2 Epidemiological Transitions

Bioarchaeological investigations of the Industrial Revolution are particularly important since understanding the health effects of urbanisation and industrialisation in the past can inform researchers about the potential threats to health in present-day developing nations undergoing these developments. Speaking in terms of evolution, humans were adapted to rural environments and so the creation of urban environments has led to adaptive biological changes (Boyden, 1972; Brüne and Hochberg, 2013a; Schell and Ulijaszek, 1999). However, as evolutionary adaptations have not occurred at the same pace as environmental changes, the movement towards urban lifestyles has resulted in negative consequences for human health (Boyden, 1972; Brüne and Hochberg, 2013a, b; Magliano *et al.*, 2013; Schell and Ulijaszek, 1999; Waldron, 1989). This was first seen in the transition from nomadic hunter-gatherer to settled agricultural societies

(1st epidemiological transition). This shift introduced novel infections and parasites due to exposure to newly-domesticated animals and increased population densities in the newly formed permanent settlements (Eshed *et al.*, 2010; Harper and Armelagos, 2013; Nicklisch *et al.*, 2012; Pearce-Duvet, 2006; Reinhard *et al.*, 2013). Many pathogens need a large enough pool of humans or animals to infect in order to survive within a location. Groups of hunter-gatherers were seldom large enough for a disease to become endemic; this only occurred after settlement into larger communities spurred by the introduction of agriculture. Additionally, these settlements increased contact between people and decreased hygiene and sanitation (Larsen, 1995). The introduction of agriculture also increased the reliance on a few food items (generally cereals that happened to lack many vital nutrients), rather than the broad range that was utilised by hunter-gatherers (Larsen, 1995; Whittle, 1999). This shift in diet led to poorer nutrition with increased frequencies of dental caries (Eshed *et al.*, 2010; Larsen, 1995; Willis and Oxenham, 2013). Increased "stress" indicators such as cribra orbitalia, porotic hyperostosis, and dental enamel hypoplasia, were also seen during this shift due to the strains placed on the body by increased exposure to pathogens and a limited diet (Eshed *et al.*, 2010; Larsen, 1995; Starling and Stock, 2007).

The second transition occurred at the start of the Industrial Revolution with increased urbanisation and more developed industry, which further compromised health and well-being (Budnik and Liczbińska, 2006; Lewis, 2002; Lewis and Gowland, 2007). Documentary evidence shows higher rates of mortality (infant and adult), a decline in stature, and complaints about air pollution (Brimblecombe, 1978, 1987; de Montluzin, 2002; Johnson and Nicholas, 1997; Korzeniewicz, 1985; Steckel and Floud, 1997). Bioarchaeological studies have proven an increase in infant mortality associated with urbanisation and industrialisation (Budnik and Liczbińska, 2006; Lewis, 2002; Lewis and Gowland, 2007). Furthermore, it has been noted that increased prevalence rates for certain diseases such as rickets (caused by a lack of vitamin D) and scurvy (caused by a lack of vitamin C) are present in these archaeological populations due to poor nutrition and thick air pollution

blocking out the sunlight needed for the body to synthesise vitamin D (Lewis, 2002; Mays, 1999; Waldron, 1989).

However, not all urban settlements, whether from the past or today, can be said to be fit in the same category: each city or town has its own unique set of potential detrimental factors and microclimate (Gianicolo *et al.*, 2012; Li *et al.*, 2013; McMichael, 1999; Schell and Ulijaszek, 1999). Even within an urban centre, individuals are exposed to varying health risk factors, particularly due to their socioeconomic standing. For example, lower socioeconomic classes are often more greatly exposed to pollutants and poor diet (Acuña-González *et al.*, 2011; Agha *et al.*, 2013; Czerwinski, 1999; Dowler, 1999; Ebela *et al.*, 2011; Fan *et al.*, 2013; Schell and Ulijaszek, 1999). Observed differing patterns of disease in post-medieval skeletal remains may therefore be useful in differentiating between socioeconomic groups. Post-medieval historical documentation can only give us glimpses of life in the past, but so too can skeletal remains. It is possible to gain a fuller picture of living conditions in the post-medieval period, particularly of Northeast England, by combining the two sources of information, as this thesis attempts to do (Bayley and Crossley, 2004; Nijhof, 2004; Petts and Gerrard, 2006).

1.3 Research Aims, Objectives, and Hypothesis

Following the rationale above, the aims of the research were:

- To assess the impact of teratogenic agents on 18th and 19th century populations in Northeast England;
- To consider the importance of settlement type (urban/rural) on the frequencies of congenital defects; and
- To compare the prevalence of congenital defects between contemporaneous populations in the two contexts.

The objectives were:

- To generate a population study of congenital defects in 18th and 19th century Northeast England; and
- To provide a better understanding of the effects of health and living environment on the occurrence of congenital defects.

The hypothesis tested was that congenital defects were more common in urban populations rather than populations from rural locations due to the detrimental living conditions associated with the urban, industrial post-medieval period. To test the proposed hypothesis, this study explores the frequency of congenital defects in skeletal remains from burials in cemeteries associated with two coastal urban centres and two inland rural sites in Northeast England. It is suggested that the rates of congenital defects should be similar between the urban populations if these anomalies are caused by factors due to increased urbanisation and industrialisation. The rural sites serve as "neutral" populations and people interred there should show fewer anomalies than those buried in the urban sites.

This study concentrates on the conurbation of Newcastle-upon-Tyne and its surrounding towns, as it was one of the largest urban areas of England during the early Industrial Revolution (Borsay, 1989; Ellis, 2000, 2001a; Langton, 2000; Walton, 2000). Common modern day usage tends to view the Northeast as separate from Yorkshire, but during this period Newcastle's wide rural hinterland extended into northern Yorkshire. Therefore, for the purposes of this study, Northeast England is defined as the areas of Northumberland, Durham, and North Yorkshire. The sites are dated largely to the 18th and 19th centuries (see Section 1.4). To the author's knowledge, this is the first study undertaken to compare congenital defects in urban post-medieval populations in England, as for example, Barnes (1994) studied prehistoric skeletons from American sites, and Sture (2001) compared rural with urban sites in medieval England.

1.4 Study Sites

Settlements in the post-medieval period can be classified as urban, based largely on population size (generally a population over 5,000 though some historians use population size as low as 2,500) and an economy based on industries other than agriculture (Floud and Harris, 1997; Slack, 2000; Wrigley, 2004). Rural settlements would therefore have a smaller population and largely rely on agriculture for their means of support, although other small-scale industries were still often practised. Most bioarchaeological research undertaken for this time period has largely focused on London (*e.g.* Brickley *et al.*, 1999; Miles *et al.*, 2008a, b; Waldron, 1993) with a few studies in other cities/towns in the south of England (*e.g.* Mays and Keepax, 2006; Start and Kirk, 1998) and the midlands (*e.g.* Brickley *et al.*, 2006a; Waldron and Rodwell, 2007). By contrast, very little work seems to have been completed in the north to date (Cherryson *et al.*, 2012). In order to help alleviate this disparity, two urban cemetery sites from North and South Shields, towns with largely industrial based economies, were chosen (Hodgson, 1903; Moffat and Rosie, 2005; Openshaw, 1978; Thornborrow, 1988; Warren, 1980). The rural sites, Fewston and Wharram Percy, were also selected due to their geographical proximity to the urban sites. The four study sites are introduced below but are discussed in more detail in Sections 2.3 and 3.1.

In considering the hypothesis to be tested, and aims and objectives of this research, the first urban site is located in North Shields, North Tyneside near the mouth of the River Tyne, just downstream from Newcastle-upon-Tyne. The main industries of the town in the post-medieval period were salt and glass making, shipbuilding, and maritime trade (Ellis, 2001b; Linsley, 1992; Reid, 1845a; Simpson, 1988; Thornborrow, 1988). During the 18th century, the population was estimated at 7,000 (Walton, 2000). This grew in the 19th century with the population reaching 9,454 in 1821 (North Shields combined with Tynemouth) before rising to about 25,808 in the 1840s (Manders and Potts, 2001; Reid, 1845a). The community represented in the Quaker cemetery at North Shields consisted largely of artisans and middle class individuals (MF176). The second urban site, South Shields, South

Tyneside, is located directly across the River Tyne from North Shields. Many similarities existed between the two towns leading at least one anonymous writer of the 19th century to comment on how the individuals in each town were virtually identical in their occupations and habits (Manders and Potts, 2001). Common industries included salt, glass, and chemical production as well as shipbuilding and maritime trading (Campbell, 1968; Hodgson, 1903; Hood Coulthard, 1960; MF831; Thornborrow, 1968, 1988). South Shields was one of the fifty largest towns in Britain in 1801 with a population of 11,011 (Hodgson, 1903; Langton, 2000). The population rose steadily throughout the 19th century, with census records recording a population of 16,503 for 1821, 18,756 for 1831, 23,072 for 1841, and 28,974 for 1851 (Hodgson, 1903; Hood Coulthard, 1959; Manders and Potts, 2001; Thornborrow, 1971a).

The first site from a rural context is located in the parish of Fewston, North Yorkshire. The parish consisted of the villages of Fewston, Blubberhouses, Clifton, Norwood, Thruscross, and Timble Great. The population of the parish in 1811 was 2,178 which decreased to 1,989 in 1821 (Baines, 1822). Agriculture was the main occupation of people who lived there in this period, with textiles and small crafts such as carpentry and blacksmithing employing smaller numbers (Baines, 1822; Harker, 1988; Parkinson, 1899; PR/FEW 1/16, 1/17). The second rural site is the cemetery of the parish church of Wharram Percy, North Yorkshire (*N.B.* Wharram Percy was historically in the East Riding of Yorkshire. For the sake of consistancy for all sites, the modern county division of North Yorkshire will be used throughout this thesis to indicate the location of this village.). The village of Wharram Percy was depopulated in the medieval period, with only a farmstead, a rectory and a few other residential buildings existing in the post-medieval period. The church remained the parish church for the surrounding vicinity until the 19th century for the villages of Raisthorpe, Birdsall, Burdale, Thixendale, and Towthorpe. The population of the parish was recorded as 345 in 1811 and then fell to 336 in 1821 (Baines, 1823). The main occupation of the people in the parish was agriculture with some small scale craft-work such as stone masonry and shoemaking taking place (Baines, 1823; PR/WP 3). The individuals of the parish were largely poor but

there were some wealthy enough to afford high-status burials inside the church (Beresford and Hurst, 1990; Mays *et al.*, 2008; Pickles, 1996).

1.5 Thesis Structure

To meet the aims and objectives set out in Section 1.3, the structure of the thesis is as follows:

- Chapter 2 is a five part literature review. Teratogenic agents discovered through modern clinical research and which potentially existed in the post-medieval period are considered in Section 2.2. Section 2.3 illustrates the living conditions experienced in the post-medieval period in Britain. The research focuses on Northeast England and pays special attention to the towns or parishes from which the cemetery populations derive. The congenital defects examined for in this study are described in Section 2.4. "Stress" indicators recorded in an effort to demonstrate poor living conditions are detailed in Section 2.5. To complete the chapter, Section 2.6 examines the bioarchaeological literature for reports of congenital defects previously found at post-medieval archaeological sites in Britain.

- Chapter 3 describes the materials and methods used for this study. The cemetery contexts are described, along with a brief account of the archaeological excavations. The congenital defects recorded for this study are listed along with information on the diagnosis of each defect. A similar treatment is provided for the "stress" indicators used in this study.

- Chapter 4 provides the results of the bioarchaeological assessment of the skeletal remains.

- Chapter 5 discusses these results in relation to potential teratogenic agents in the living environments of these populations. The results are then compared to other contemporaneous populations to place the data in a

broader context. This chapter also includes discussion of the limitations and complications of studying congenital defects in archaeological populations.

- Chapter 6 draws together the conclusions of the study. It restates the overall findings of the study in relation to the hypothesis, aims, and objectives detailed in Section 1.3, provides limitations to the study, and gives recommendations of future opportunities for work.

CHAPTER 2
LITERATURE REVIEW

2.1 Introduction

In order to assess the validity of the study hypothesis (Section 1.3), it is necessary to investigate the information that is currently available on congenital defects and living conditions in the past. This chapter is a five part literature review designed to explore the existing clinical, bioarchaeological, and historical documentation for insight into these topics. First, the causes of congenital defects are investigated through modern clinical literature in Section 2.2, with particular emphasis on environmental factors (teratogens) that may have affected the skeletons of 18th and 19th British populations. Next, historical documentation is explored for evidence of living environments found at the four study sites (see Section 1.4) as these conditions may have been detrimental to health and led to the formation of congenital defects. This is followed by two sections on the congenital defects (Section 2.4) and "stress" indicators (Section 2.5) observed in the bioarchaeological assessment of this study. For both sections, modern clinical and bioarchaeological texts are consulted to explain the definition, development, and appearance of each trait. Finally, the bioarchaeological literature is examined for evidence of congenital defects in the 18th and 19th centuries in Britain to demonstrate that congenital defects can be seen for this period.

2.2 Teratogen Literature Review

'To see pattern and extremes in variation is to glimpse the world as seen by natural selection. It is not a world of uniformly tiny, mutationally based, or exclusively quantitative variants. Rather it is one full of recurrent developmental anomalies that vary in accord with the genetic makeup of individuals and also with their environmental circumstances.'
(West-Eberhard, 2003:208)

2.2.1 Causes of Congenital Defects

Congenital defects arise due to intrinsic factors, extrinsic factors, or a combination of the two (Fan *et al.*, 2013; Schoenwolf *et al.*, 2009; WHO, 2012). Intrinsic factors can include genetics, age, sex, ethnicity, and immunity (Czerwinski, 1999). Genetic factors are inherited single gene disorders, inherited chromosomal disorders, or unique mutations occurring at conception (Barnes, 1994, 2012b). Extrinsic or environmental factors which can include malnutrition, infectious disease, and heavy metals, and act after conception to alter the development of the fœtus (Aufderheide and Rodríguez-Martín, 1998; Czeizel, 2008; Holmes, 2011; Ortner, 2003). However, genetic factors can interfere with the expression and severity of a defect caused by environmental factors (Schoenwolf *et al.*, 2009). Alternatively, environmental factors can affect genes through allele inactivation or deletion and mutation of methyl transfer enzymes, ultimately leading to the formation of defects (Friedman, 2011; Kubota, 2008). The combination of genetic and environmental factors is known as epigenetic interaction (Barnes, 1994, 2012a, b; Friedman, 2011). In epidemiological principle, two fœtuses exposed to the same level of a teratogen at the same stage of development can have very different outcomes due to their genetic makeup. The term teratogen, as defined in Section 1.1.1, applies primarily to extrinsic agents but the term can also be applied to include genetic abnormalities (Lancaster, 2011). For the purpose of this research and to avoid confusion, teratogen will be applied solely to extrinsic factors that cause defects.

For an agent to be considered teratogenic in humans it has to have some if not all of the following characteristics (Barnes, 1994; Holmes, 2011; Gluckman and Hanson, 2005; Schoenwolf *et al.*, 2009):

1. There should be a demonstrable significant increase in a specific outcome (*i.e.* defect) after exposure (Holmes, 2011). A specific defect should occur more commonly in infants of mothers exposed to a particular factor than those who were not;
2. It must be demonstrated that presence of the defect makes sense biologically. It should be possible to show in the biological pathways of fœtal development where the insult, or event causing damage, takes place (Barnes, 1994; Holmes, 2011);
3. There should be a relationship between the amount of exposure and the teratogenic outcome. Generally, the greater the exposure the more likely a defect will arise and the more severe it will be (Holmes, 2011);
4. A threshold should exist, where there is a level of exposure below which no harm is caused (Gluckman and Hanson, 2005; Holmes, 2011; Schoenwolf *et al.*, 2009);
5. A period of greatest sensitivity in the pregnancy can be seen where exposure is more harmful (Acuña-González *et al.*, 2011). This period is usually in the first trimester but can occur at any time during pregnancy (Czeizel, 2008). The formation of a fœtus is ordered with shaping of various organ systems and body parts occurring during specific days or weeks of a pregnancy. For example, a teratogen that only affects the kidneys will have no effect on a fœtus if the kidneys are not developing at the time of exposure;
6. There is a genetically more susceptible group of individuals (Holmes, 2011). Due to epigenetic interactions (described previously in this section), some individuals will be more greatly affected by the exposure of a teratogen due to their genetic predisposition. Two pregnant women may be exposed to the same level of a teratogen at the same stage in pregnancy and yet there may be different outcomes in terms of defects for the infants due to their genetic susceptibility or lack thereof; and
7. It must be demonstrated that an animal model exists where exposure to an agent through the same route (*e.g.* oral, respiratory, etc.) as that

experienced by humans causes harmful effects (Holmes, 2011). Many of the findings in Section 2.2.2 below are based on animal models that have been interpolated as causing similar effects in humans.

2.2.2 Teratogens

There are numerous teratogens that affect a wide variety of organs and organ systems. Human teratogens can be divided into seven categories: (i) drugs/pharmaceuticals, (ii) maternal conditions (*e.g.* diabetes, excessive alcohol consumption), (iii) intrauterine infections, (iv) heavy metals, (v) radiation, (vi) medical procedures during pregnancy (*e.g.* chorionic villus sampling), and (vii) other (*e.g.* heat, sniffing petrol/gasoline) (Holmes, 2011; Wilson, 1973). Many teratogens are related to modern life such as nuclear radiation and modern pharmaceuticals (*e.g.* thalidomide, antidepressants, anti-thyroid medications, etc.) so would not be a concern in an archaeological context (Andersen *et al.*, 2013; Connor and Ferguson-Smith, 1997; Gianicolo *et al.*, 2012; ver der Lugt *et al.*, 2012; Wilson, 1973). The discussion of specific teratogens here will focus on those that may have been present in post-medieval British populations and would have had the potential to affect the skeleton. As such, only three categories of teratogens will be examined: maternal conditions, intrauterine infections, and heavy metals, the other categories are associated with modern life. Table 2.1 provides a summary of the teratogens discussed in the next sections and the congenital defects they cause. Congenital defects from the table that were observed in this study are defined and discussed in Section 2.4.

Category	Teratogen	Defects
Maternal Conditions	Diabetes	neural tube defects, absent lumbar or sacral elements, femoral head aplasia, heart defects, brain defects
	Hypervitaminosis A	craniosynostosis, calvarium hypoplasia, mandible malformations, cleft palate, craniofacial defects, supernumerary vertebrae, absent vertebrae, premature long bone fusion, cardiovascular defects, central nervous system defects
	Zinc Deficiency	cleft lip and palate, anencephaly, neural tube defects, spina bifida, absent vertebrae, fused thoracic and lumbar vertebrae, spinal curvatures, rudimentary ribs, fused ribs, syndactyly, clubbed feet, agenesis of limbs
	Iron	spine defects, rib defects
	Manganese	domed skull, shortened long bones, dysplasia of tibial epiphyses
Infectious Disease	High Fevers	cleft lip and palate, microphthalmia, facial defects, microcephaly, neural tube defects, neurogenic limb contractures, external genital defects, external ear defects
	Rubella	microcephaly, heart defects, mental handicap
	Lymphocytic Choriomeningitis Virus	encephalitis, microcephaly, macrocephaly, hydrocephalus, neurological defects
Heavy Metals	Selenium	spinal curvatures
	Stannous Chloride	microcephaly, microphthalmia
	Benzo(a)pyrene	craniofacial defects
	Lead	fused cervical vertebrae, delayed ossification
	Arsenic	cleft lip and palate, neural tube defects, fused vertebrae, occipitalization, fused ribs
	Mercury	cleft lip and palate, encephaly, fused ribs, clubbed feet, syndactyly
Other	Hypoxia	cleft lip, craniofacial defects, neural tube defects, limb reduction defects, heart defects, brain defects
	Carbon Monoxide	microcephaly, skull defects, mandible defects, wedged vertebrae, fused vertebrae, absent vertebrae, hemivertebrae, scoliosis, fused ribs, lumbar ribs, brain damage
	Carbon Dioxide	vertebral defects, ectrodactyly, heart defects

Table 2.1: Summary of teratogens and the congenital defects they may cause. Defects in blue may affect the skeleton and be seen archaeologically.

A. Maternal Conditions

Congenital defects can be caused by conditions such as disease and malnutrition. An example of a maternal medical condition being teratogenic is diabetes. Due to the health complications of diabetes, it is possible that many females with the disease in the past would not have lived to a reproductive age, but gestational diabetes, or diabetes brought on by pregnancy, does not always require drastic medical intervention or modern medications. Hyperglycemia from diabetes can lead to neural tube defects, missing lumbar or sacral vertebral elements, aplasia of the femoral head, and heart and brain defects (Bánhidy *et al.*, 2011; Ornoy *et al.*, 2010; Wilson, 1973).

Both excesses and deficiencies of various vitamins and nutrients in the maternal diet can also be teratogenic. An excess of vitamin A (hypervitaminosis A) can cause craniosynostosis, hypoplasia of the calvarium, mandible malformations, cleft palate and other craniofacial anomalies, supernumerary and missing vertebrae, premature long bone fusion, cardiovascular defects, and central nervous system abnormalities (Abbott, 2010; Chapman, 2012; Laue *et al.*, 2011; Villenueve *et al.*, 2006). Vitamin A is found in a variety of foodstuffs including meat, particularly liver, and vegetables such as carrots, pumpkin, broccoli, and spinach (Chapman, 2012). On the deficiency front, even a relatively mild lack of zinc can lead to neural tube defects/spina bifida, missing vertebrae, fused thoracic and lumbar vertebrae, spinal curvatures, cleft lip and palate, anencephaly, rudimentary ribs, fused ribs, syndactyly (fused digits), clubbed feet, and agenesis of limbs (Hurley, 1981; Shah and Sachdev, 2001; Uriu-Adams and Keen, 2010). Other possible effects include neurobehavioural and immunological abnormalities. The WHO in 2002 listed zinc deficiency as the fifth highest risk factor for disease in developing countries (Uriu-Adams and Keen, 2010). Mild to moderate zinc deficiencies are common in modern developing countries, particularly amongst vegetarians or women with limited access to meat. Elevated levels of dietary iron in a pregnant woman may also lead to malformations of the spine and ribs (Weinberg, 2010), while manganese deficiency can cause shortened long bones, a domed skull, and dysplasia of tibial epiphyses (Hurley, 1981).

B. Infectious Disease

Maternal infectious diseases can cause a wide variety of congenital defects. High fevers from influenza, the common cold, or tonsillitis can produce neural tube defects, orofacial clefts, microphthalmia, facial anomalies, microcephaly, neurogenic limb contractures, deformities of external genitals, and external ear defects (Czeizel *et al.*, 2008; Lancaster, 2011). Fevers of over 38.5^{o}C/101.3^{o}F need to last only one day to affect fœtal development. The timing of illness determines which organ systems are affected. Specific viruses have also been implicated in causing congenital defects. Rubella, also known as German measles, can cause microcephaly, heart defects, and mental handicap (Connor and Ferguson-Smith, 1997). A second virus, lymphocytic choriomeningitis, is spread by mice through inhalation of the virus or direct contact with contaminated droppings (Barton and Mets, 2001), and infection with this virus in an expectant mother in the first or second trimester can lead to encephalitis, micro- and macrocephaly, hydrocephalus, and possible neurological abnormalities in the fœtus (Barton and Mets, 2001; Hannachi *et al.*, 2011; Rousseau *et al.*, 1997). Fungal infections are another source of defects as mycotoxins, natural contaminants produced by fungal species, are teratogenic (Köppen *et al.*, 2010; Peraica *et al.*, 1999). These moulds and toxins are usually found in tropical regions but are also seen in temperate areas as well.

C. Heavy Metals and Other

A major source of teratogens is maternal exposure to pollution and the heavy metals associated with industry. Selenium has been found to cause curvatures of the spine (scoliosis and kyphosis) and is released into the environment by the coal mining process or as a byproduct of sulphuric acid production (de Rosemond *et al.*, 2005; Flohé, 2009; Miller *et al.*, 2009). Exposure to stannous or tin chloride can lead to defects such as microcephaly and microphthalmia (Şişman, 2011). Contact with benzo(a)pyrene, a contaminant associated with urbanisation, causes craniofacial malformations (He *et al.*, 2011) while delayed ossification and fused cervical vertebrae are congenital defects

caused by maternal exposure to lead (Jacquet and Gerber, 1979). Arsenic exposure can cause neural tube defects, fused vertebrae, occipitalization, cleft lip and palate, and fused ribs (Hill *et al.*, 2008; Léonard and Lauwerys, 1980; Machado *et al.*, 1999), but the teratogenicity of arsenic in humans has been disputed (DeSesso *et al.*, 1998; Wang *et al.*, 2006). Finally, exposure to mercury, even at low levels, can lead to cleft lip and palate, encephaly, fused ribs, clubbed feet, and syndactyly (Tchounwou *et al.*, 2003). While not a heavy metal, hypoxia can be included in this category for the purposes of this study since it can be associated with heavy industry. Hypoxia, or lack of oxygen, can be the result of breathing in an atmosphere deficient in oxygen such as in smoky environments (Ornoy *et al.*, 2010). Defects associated with hypoxia in pregnancy include neural tube defects, transverse limb reduction defects, craniofacial anomalies including cleft lip, and heart and brain defects (Ornoy *et al.*, 2010; Webster and Abela, 2007). Smoky environments, if produced by coal fires, are rich in carbon monoxide (CO) and carbon dioxide (CO_2). Even if the mother is exposed only once and for just a few hours at levels tolerable to her, carbon monoxide can lead to the formation of lumbar ribs and brain damage in her unborn child (Ema *et al.*, 2010). Other abnormalities seen are wedged, fused, and missing vertebrae, hemivertebrae, scoliosis, fused ribs, microcephaly, and malformed skulls and mandibles (Loder *et al.*, 2000; Singh *et al.*, 1993). The worst defects are seen when maternal diet is protein-limited. Excess exposure to carbon dioxide has also been shown to create vertebral anomalies, ectrodactyly ("lobster claw hands"), and heart defects (Ema *et al.*, 2010).

2.2.3 Teratogen Conclusions

As this section has demonstrated, a wide assortment of congenital defects can be caused by a variety of environmental (extrinsic) factors. Many of these defects affect the skeleton and as such may be seen archaeologically. Next, Section 2.3 will examine the living conditions of the post-medieval period in Northeast England for evidence of teratogens and other factors detrimental to health. These poor living conditions revealed by historical documents may therefore suggest that congenital defects may have been present in the affected populations.

2.3 Historical Literature Review

'These new towns were at their most raw, untrammeled, dramatic, exciting and threatening: "great human exploits" which produced and distributed a cornucopia of goods under a shroud of infernal smoke and under conditions which visibly threatened life, health and social and political stability.'
(Walton, 2000:111)

Section 2.2 has shown a myriad of environmental teratogens that can lead to the development of congenital defects in human populations. It is therefore essential to explore the presence of these and other factors detrimental to health in the past. This can be done through the investigation of environmental and living conditions, as this will highlight potential risk factors for people from this period. The 18th and 19th centuries saw a rapid increase in urbanisation and industrialisation. Great numbers of people migrated to newly-developed or expanded towns and cities to find employment in the growing industries there (Alderman, 1986; Ellis, 2001a; Langton, 2000; Morris and Rodger, 1993; Sharpe, 2000). This sudden increase in population and industry brought very serious problems for health, but particularly affected the ability to provide good housing, effective sanitation, a sufficient and nutritious food supply, clean water, and the means to mitigate poor air quality (Belford, 2004; Cruickshank and Burton, 1990; Hey, 1986; Korzeniewicz, 1985; Moffat and Rosie, 2005; Walton, 2000). It is popularly thought that there was a strict dichotomy in the quality of the "environment" between urban towns and rural areas: urban centres were smoky, disease-infested, and overcrowded while rural villages were wholesome, healthy idylls (Ellis, 2001a; Korzeniewicz, 1985; Robinson, 1847; Sharpe, 2000; Woods, 2003). As evidence, contemporaries espoused the healthiness of individuals raised in rural settings compared to those from towns (Robinson, 1847; Sharpe, 2000). However, these eyewitness accounts were commonly produced by middle class reformers or moralists intentionally painting areas occupied by the working classes in an unwholesome light (Belford, 2001; Ellis, 2001a; Johnson, 1993; Mayne and Murray, 2001; Ross, 2001) so that when investigating the actual circumstances experienced by people of the period, this dichotomy does not seem to be supported.

This section explores historical and archaeological documentation for the living conditions experienced in both urban and rural environments in the post-medieval period. Factors examined include the quality of housing, levels of sanitation, quality of the water supply, the occupations in which residents were employed, air quality, evidence of the diet people consumed, and access to health care. This is because any one or combination of these factors may have been responsible for the occurrence of congenital defects within these populations. Particular care has been taken to concentrate on the four study sites described in Section 1.4. Information relevant to North and South Shields is discussed in the urban sections while the rural sections contain information for the parishes of Fewston and Wharram Percy as well as the wider area of North Yorkshire.

2.3.1 Housing

'In a low, damp, dirty, ill-ventilated, miserable hovel, kept by the most filthy people I ever beheld...' Gilbert Ward, medical officer for Tynemouth
(Chadwick, 1842:415)

A. Urban

Housing in post-medieval towns, particularly that of the rapidly expanding northern industrial and manufacturing centres, was notoriously unsanitary and overcrowded. The houses in North and South Shields were no exception. Narrow, crowded streets, alleyways, and courts contained the dense population (Berry, 2010; Garson, 1992; Grundy *et al.*, 1992; Reid, 1845a, b). Low-cost housing was generally available for those who needed it but was guaranteed to be low-grade, damp, and poorly-ventilated (Chadwick, 1842; Woodward, 1995). In fact, words commonly used to describe houses in North and South Shields included: dilapidated, unwholesome, ill-ventilated, filthy, and miserable (Chadwick, 1842; Reid, 1845a, b). The specific details of housing in these towns that led to such remarks are investigated below.

The densely packed houses of North and South Shields arose out of spatial building restrictions. North Shields originated along Low Street, running parallel to the River Tyne (Figure 2.1) (Garson, 1992; Grundy *et al.*, 1992; Simpson, 1988).

Expansion to the south was blocked by the river and to the north by steep hills. Space was at such a premium that houses were built very close to the river's edge – so close in fact that they were supported by slanting wooden beams coming out of the river (Garson, 1992). By 1760, expansion northwards to the top of the hills was necessitated by the growth and abundance of the population and its industries (Garson, 1992; Grundy *et al.*, 1992). The hills were too steep to construct roads and therefore stairways were constructed between the old and new towns. Tenements, or subdivided houses, filled with the working poor sprang up along these stairs (Reid, 1845a; Simpson, 1988). The expansion northwards also created space for the better housing of the middle and upper classes. The first houses were built in 1763 on the hilltops at Dockwray Square, largely by ship-owners (Garson, 1992). A second housing development started at Milburne Place around the same time when the aristocrats of North Shields took to building substantial homes (Simpson, 1988). These new houses had lavatories and ash-pits, both necessary sanitation features that were almost completely lacking in the lower town (Reid, 1845a). However, even though these houses were built and lived in by the middle and upper classes, the new developments suffered from many of the same sanitary problems of drainage and disposal of sewerage that they had tried to leave behind in the old town (Simpson, 1988). Indeed, some of these issues were not solved until the 20th century.

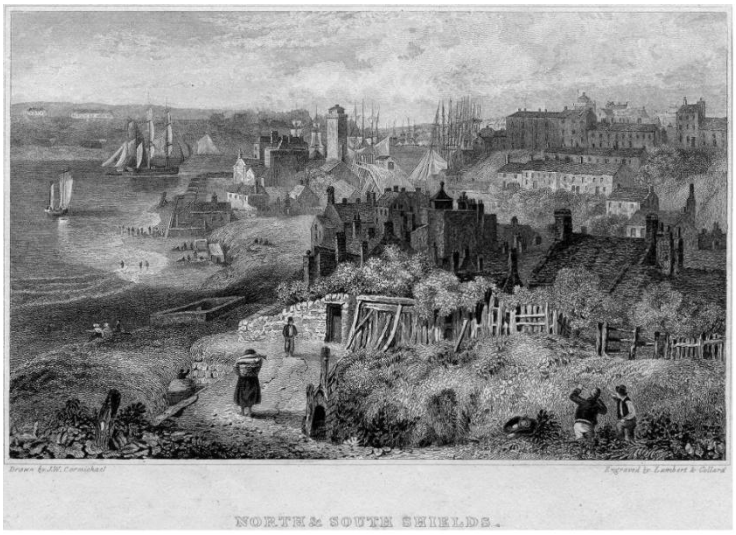

Figure 2.1: A print showing the closely built houses of North Shields before 1847 (Pictures in Print, 2012).

The patterns in town expansion at South Shields were similar to that seen at North Shields. Here, South Shields had largely confined itself to the banks of the River Tyne as homes were built close to the industrial areas set up on the riverside (Figure 2.2) (Openshaw, 1978). Expansion of the town started in the 1760s, eventually developing westwards. By the 19th century, the upper classes had largely moved out of the town and away from the unsanitary, cramped conditions (Foster, 1975).

Figure 2.2: A print of South Shields *c.* 19th century showing the houses closely built together along the riverside (Pictures in Print, 2012).

The confined space due to the geographical layout of the towns was mirrored inside the houses themselves. Nationally (and in Northeast England), starting in the 16th century with the rise in the population, combined with increased movement into urban areas, the existing housing stock started to be exhausted (Ayers and Roney, 1993; Moffat and Rosie, 2005; Reed, 2000; Woodward, 1995). Houses and cottages originally built for single family occupation were subdivided with one family occupying one room or storey of a building (Reid, 1845a; Woodward, 1995). At North Shields, each tenement block housed an average of five families (Reid, 1845a), with the average room size measuring 15 by 15 feet and being inhabited by four individuals. This population density slightly declined late into the 19th century with census data from 1851 to 1901 showing an average of ten individuals per building, although this was still high compared to the seven individuals per building in higher class Tynemouth (Atkinson, 1989).

At South Shields, the number of families per house ranged from one to seventeen with an average of three (Reid, 1845b). The average room housed three individuals and was 12 feet by 14 feet with a ceiling height of seven and a half feet. The apartments in these subdivided houses were generally connected by a single staircase (Reid, 1845a, b). As these houses were not provided with lavatories of any kind, the staircase often served as a storage facility for pails holding each household's refuse and "filth" awaiting the daily visit of scavengers, who were individuals paid to collect rubbish (Reid, 1845a). Some houses built against hillsides in South Shields were spared this unsanitary custom as the raised ground at the back of the buildings allowed for separate entrances for the upper and lower stories (Reid, 1845b).

Houses were typically two or three stories and constructed of brick or stone with tiled roofs (Green, 2003; Grundy and McCombie, 1992; Reid, 1845b; Woodward, 1995). In both North and South Shields, few cellar dwellings existed (Reid, 1845a, b). At South Shields cellar dwellings were described as being well lit and having fire places but suffering from drainage and ventilation problems. Few houses in North Shields were built back to back while many more were constructed in this way in South Shields (Hodgson, 1903; Reid, 1845a, b). Back-to-back houses severely increased the population density for a given area, limited the amount of outdoor space available to tenants, put strain on water supplies and sewers (when present), and compromised the amount of available light and air.

At this time, few, if any, building regulations existed. Buildings destined to be "tenemented" or subdivided were often poorly constructed in undesirable locations (Burnett, 1978; Hodgson, 1903; Reid, 1845a, b). In the case of South Shields, tenement houses were built on the refuse piles of the glassworks (Hood Coulthard, 1960). As the refuse largely consisted of under fired coals, fires were a constant concern (Hodgson, 1903). Tenements were generally poorly looked after by owners, with the buildings commonly falling into disrepair and found to be in an 'objectionable state of repair and condition' (Reid, 1845a:182).

The apartments of the poor had few windows (Reid, 1845a; Robinson, 1847). Stagnant, unpleasant-smelling air believed to be the major cause of disease

under the miasma theory of the time so the lack of ventilation was a major contemporary concern. These dwelling places were commonly referred to as close, unwholesome, and bad (Reid, 1845a, b). Windows would have been necessary to ventilate the smoky interiors and the circulation of air would have helped to reduce dampness, the spread of infectious disease and conditions associated with poor air quality.

Despite the negativity discussed about housing conditions in North and South Shields, there were some positives. Due to the large-scale coal shipping that occurred in these port towns, coal was an abundant and cheap fuel, often allowing for homes to be well heated (Reid, 1845a, b). Yeomen, craftsmen, and many workers in industries present in North and South Shields would also have been able to afford slightly better accommodation than would generally have been expected (Green, 2006; Woodward, 1995). The terraced housing commonly built on Tyneside and Wearside starting in the late 17th century to house some of these better-off workers may have provided better accommodation than that inhabited by agricultural labourers (*ibid.*).

B. Rural

In the rural areas of Yorkshire, little changed in the way of housing architecture from the late medieval period to the first century or two of the post-medieval period. In fact, many of the inhabited houses may have originated during the 16th century or earlier (Giles, 2006; Jennings, 1967). Starting in the second half of the 17th century, there was a marked rise in standards of living within the yeoman class due to increasing wealth and aspirations, leading to extensive rebuilding of houses (Jennings, 1967). The introduction of a fire hood rather than an open hearth and the addition of a second fireplace were part of this rebuilding (Giles, 2006). The 19th century saw a further rise in the standards of housing in the region.

The humblest building materials were mud or chalkstone. The use of chalk was particularly common in the East Riding and Wolds of Yorkshire and was generally of poor quality, being prone to severe weathering (Beresford and Hurst,

1990; Hey, 1986; Neave and Neave, 2006). The better constructed cottages were cruck-framed (timber supports) with masonry walls for the lower half and lath and plaster or wattle and daub for the upper (Beresford and Hurst, 1990; Jennings, 1967; Mercer, 1975; Pallister and Wrathmell, 1990). The thickness and construction quality of the masonry portion of the walls were dependent on socioeconomic standing (Pallister and Wrathmell, 1990). Regardless of the building materials of the walls, all houses had thatched roofs (Hey, 1986; Jennings, 1967). By 1680, there was a move towards local stone and brick as the favoured building materials, with slate tiles or pantile made from fired clay for all roofs regardless of socioeconomic level (Figure 2.3) (Hey, 1986; Neave and Neave, 2006). However, the pantiles could be easily lifted by the wind, allowing rain to be absorbed into the fabric of the walls unless a bottom course of slates was laid first. It was not until the later 18th or 19th centuries that pantiles became widely accepted (Hey, 1986).

Figure 2.3: Fewston village in a photograph *c.* 1900-1910 (Alred, 1997).

In the middle of the 17th century, the typical house layout consisted of a central living room (the "house") containing the hearth with a separate parlour for sleeping in (Giles, 2006; Hey, 1986; Mercer, 1975; Neave, 2010; Neave and Neave, 2006). Upstairs, one or more chambers would be located for additional sleeping quarters (children and servants) and storage. The entrance to many homes included two or more steps down into the lower storey, making the ground floor resemble that of a cellar (Hey, 1986; Jennings, 1967). Most homes only had the one hearth

with a wooden chimney above, located in the “house”, leaving the sleeping rooms unheated (Giles, 2006; Jennings, 1967). About 62% of houses in rural areas had only one hearth compared to about 37% of houses in the towns of Yorkshire (Hey, 1986). The number of hearths in rural areas increased, with hearths sited in the parlour, and even upstairs in grander homes as socioeconomic status increased (Figure 2.4). Additionally, the building material of chimneys changed from wood to stone. However, the Hearth Tax, a tax based on the number of fireplaces in a home, may have led some households, regardless of socioeconomic status, to retain just the one hearth although the high costs of fuel and relative poverty were the more likely driving forces behind the number of hearths (Hey, 1986, 2006; Neave and Neave, 2006). According to hearth tax data from 1670-1674, the most common number of hearths was one (14 out of 31 homes) and the highest number reported for any house was three (Bell and Beresford, 1987). The most common fuels of the time were turves (peat) and ling (heather), with the occasional use of coal and, in poverty stricken times, cassons (dried cow excrement) (Harker, 1988; Hey, 1986; Jennings, 1967; Neave and Neave, 2006). A common feature of houses in Yorkshire, particularly in and around the West Riding, was their role as accommodation for both humans and animals at least until the 17th or 18th century (Beresford and Hurst, 1990; Hey, 1986; Hoskins, 1953; Jennings, 1967; Neave, 2010). While the animal byres and the human living areas were commonly separated by a passageway and/or screen, they did at least share a roof (Beresford and Hurst, 1990; Neave, 2010; Hey, 1986; Pallister and Wrathmell, 1990).

Figure 2.4: The 18th century vicarage at Wharram Percy with a thatched roof and three chimneys (Beresford and Hurst, 1990). On the ground floor were three rooms and a pantry with four rooms on the floor above.

The number of rooms, heated or unheated, increased or decreased with socioeconomic status. In prosperous yeoman households six was the most common number of rooms (Jennings, 1967). Cooking was removed from the "house" to a separate kitchen room with its own hearth or, particularly for those in cruck-framed houses, placed by an oven house outside the cottage (Giles, 2006; Jennings, 1967). The cottages of the agricultural labourer, by contrast, commonly had only one room (Giles, 2006; Hey, 1986; Jennings, 1967; Neave and Neave, 2006), with the interior lacking a ceiling and having a dirt/clay floor (Hey, 1986). Few, if any, windows were found in these houses. Sash glass windows were introduced amongst the wealthy in the 1680s and slowly filtered down through the classes (Hey, 1986; Jennings, 1967). The individuals living in the one room cottages would have been unlikely to afford glass windows, instead having small openings filled with lattice works or waxed linen with shutters (Jennings, 1967). This standard of housing amongst the poor was still recorded in the 19th century (*ibid.*). However, class did not entirely protect individuals, as even those in the "middling classes" were described as living in cramped accommodation in the latter half of the 18th century (Jennings, 1967). On the whole, throughout the post-medieval period the houses of rural Yorkshire were described as small, dark, and poorly-heated (Neave and Neave, 2006).

2.3.2 Sanitation and Water

'The inhabitants are generally remarkably sober, industrious, and orderly; but are in much need of improved ideas as to domestic cleanliness and comfort.'
(Reid, 1845b:184)

'The dyeing houses, scouring shops, and places where they used this water, emitted the water again tinged with the drugs of the dyeing vat and with the oil, the soap, the tallow, and other ingredients used by the clothiers in dressing and scouring.' Daniel Defoe, 1720s
(Singleton, 1970:21-22)

A. Urban

North and South Shields both suffered greatly from a lack of sanitation. It was only after a smallpox epidemic in 1870-1871 that sanitary conditions were to any great extent improved in South Shields (Hodgson, 1903). A writer of the 19th century stated that 'swine wallowed unmolested in the unwholesome sumpholes of the public thoroughfares, foul water and filthy offal were thrown with impunity upon the streets' (Hodgson, 1903:149). The streets were lined with rubbish and turned into muddy quagmires in the rain (Hood Coulthard, 1959, 1960; Reid, 1845a).

The Select Vestry of South Shields was responsible for looking after the sanitary conditions of the town. After an outbreak of fever in 1790, the Vestry hired scavengers to clean the streets but it does not seem that a regular system of street-cleaning was set up until 1849 (Hodgson, 1903). Prior to that time, the Town Commission employed as many daily scavengers to clear streets as they could afford (Hodgson, 1903; Reid, 1845b). Cleaning of the main streets was prioritized but courts and alleys were cleaned weekly. By the 1840s, North Shields employed four men to clean the streets, hiring extra assistance as needed, but some courts and alleys inhabited by the poor were often inaccessible to the dust-carts and left in a filthy state (Reid, 1845a). Few houses in North and South Shields were provided with dust-bins, and instead the rubbish was collected by dust-carts travelling through the streets daily (Addyman, 1989; Reid, 1845a, b). Each household's refuse was stored in buckets on the landings of the communal staircase in tenemented

houses (Reid, 1845a). In North Shields the refuse was deposited about a quarter of a mile from the town and sold as manure to cover the costs of removal (Reid, 1845a). South Shields provided four public places within the town for depositing rubbish and here too it was sold for manure (Reid, 1845b).

Many of the houses in North and South Shields lacked privies, or lavatories (Reid, 1845a, b; Hodgson, 1903). While South Shields had three public privies, North Shields had none, the lack of which proved a 'fertile source of street nuisance and outrage against common decency' (Reid, 1845a:182). In the absence of privies, people were forced to use the streets or the buckets used for refuse and stored in common stairwells. Where privies did exist, there was a variety of emptying methods. A few emptied directly into the River Tyne and a few more emptied into drains, but the majority emptied into ash-middens or pits (Reid, 1845a, b). Neighbouring farmers "cleansed" these middens, collecting the night soil for use as manure. In the upper and middle class areas of North Shields, the privies emptied into cesspools that were not covered or properly drained, leading to many complaints over the stench (Reid, 1845a).

Drainage and sewer systems were another major concern. Even the upper and middle classes in newly built-up areas of North Shields were not immune to deficient drainage and absent sewers (Reid, 1845a; Simpson, 1988). The natural positions of North and South Shields should have been beneficial to efficient drainage, with many of the streets sloping down towards the river and high tide reaching the edge of town (Reid, 1845a, b; Hodgson, 1903). Unfortunately, drains and sewers were seldom positioned in these streets to facilitate drainage. Only about a quarter of streets in North Shields had any sort of drain or sewer (Reid, 1845a). Refuse accumulated at the bottoms of inclines rather than being funneled towards drains or the river. In North Shields, pipes had even been installed for the cleansing of streets, but by 1845 had never been used (*ibid.*). Some houses did have drains but these were not cleaned by water, and instead were positioned so gravity would clear them (Reid, 1845a, b). This imperfect system led to blocking of the drains and many complaints over the smells emitted into houses. As the majority of

houses did not contain a drain, much of the refuse water of the household was thrown into the street. While some of this liquid was able to flow into watercourses, stagnant pools and open drains could be found in most streets and near the houses of the poor. The runoff from these streets and pools would have affected the bodies of water in the vicinity, making the water unfit for use (Moffat and Rosie, 2005). An improved sewer system in South Shields was not started until 1856, although this plan was not adopted across the entire town until 1862 (Hodgson, 1903).

From early in the post-medieval period, all towns in the North East had public water taps supplied by lead pipes (Atkinson, 1989). Both North and South Shields suffered from inadequate and/or poor quality supplies of water. North Shields was supplied by five springs arising from limestone and in 1845, of the 4,000 houses in the borough, only 233 (those of the upper classes), had piped water (Reid, 1845a). The poorer classes were supplied with water through water-carts or standing pipes with dozens of homes typically supplied by one tap (Simpson, 1988). Houses with piped water paid 18s to 30s per year for an unlimited quantity while those collecting from pipes or water-carts paid one farthing for a skeel (about six and a half gallons) (Reid, 1845a). The picture in South Shields is rather similar to that of North Shields. The town was supplied by three wells through limestone (Reid, 1845b). However, the quality of this water was suspect. The water for the wells was collected in two reservoirs that were polluted with vegetable matter and animal waste as several piggeries drained into it (Hodgson, 1903). The South Shields Water Company (which was in charge of these water supplies) was taken over in 1852 by the Sunderland Water Company because they had made no effort in attempting to address the quality and quantity of water available to the town. Of the 3,911 houses in 1845, 180 had piped water, 977 collected their water from stand-pipes, and the remaining 2,754 relied on water-carts or their own supply/well (Hodgson, 1903; Reid, 1845b). The charge for water from one of the twenty-four pipes of the town was 1.5d or 2d per week and the charge from the water-carts was one halfpenny per skeel. Water piped directly to homes was available night and day but the water was turned off at the public pipes at 3pm each day (*ibid.*). To

summarise this information, at both North and South Shields, the poor were charged more for their limited supply of water (Atkinson, 1989; Hodgson, 1903; Reid, 1845a, b; Robinson, 1847).

The poor of urban centres were generally thought of as filthy and unsanitary by their contemporaries, and those of North and South Shields were no exception (Chadwick, 1842; Reid, 1845a, b; Robinson, 1847). Industries using large amounts of coal led to a blackened sooty appearance of employees and did little to prevent this belief (Reid, 1845b). The Duke of Wellington's habit of bathing daily had popularised it amongst the upper and middle classes but this had not been adopted by the working masses (Atkinson, 1989). When South Shields issued an inquiry as to how best to reduce the number of vagrants in the town, the guardians of the Sunderland Poor-House recommended washing them (Hood Coulthard, 1959). Since instituting a requirement that everyone must bathe before being given food at the workhouse, the average number of people applying for aid had fallen from 119 to five per week. In reality, the expense of soap and water and an inadequate supply of water were more likely stumbling blocks for cleanliness than the unwillingness of the people (Reid, 1845b; Robinson, 1847). By 1850, the best the South Shields Water Company could supply was thirteen gallons per person per day for use domestically, while the water supplied to industry did not meet demand (Hodgson, 1903). By comparison, modern Britains use about 33 gallons of water per day, although this is perhaps not a true comparison due to the use of modern appliances (*e.g.* dishwasher, washing machine) today (Environment Agency, 2013). Additionally, at both North and South Shields, the populations were undersupplied with wash-houses. South Shields had only two, both located outside of the town, and used by women who made their living washing the clothes of wealthier residents (Reid, 1845b). Instead, many people resorted to creeks and other bodies of water for bathing (Thornborrow, 1968).

B. Rural

Very little seems to have been written about sanitation or access to water in rural areas of Britain in the post-medieval period. However, poor sanitation and

water quality were certainly not confined to urban areas. A local medical officer in North Yorkshire in the late 19th century reported that 'the attractive exteriors of the houses...concealed "some of the most vile and filthy places"' (Jennings, 1967:362). Improvements to sanitation were slow to come and did not start until the 19th century, much as in urban areas. In North Yorkshire most families obtained their water from springs or other nearby bodies of water, although some did have access to wells or pumps (Alred, 2001; Beresford and Hurst, 1990; Harker, 1988; Hey, 1986). Even into the 20th century not every house had access to water piped directly to their homes, with the residents still relying on springs and other sources (Alred, 2001; Harker, 1988). Lakes were often stagnant, but springs could deliver good quality water. In some areas, water supplies were plentiful but located too far from the houses. People would instead collect water from open watercourses in the street for washing, but this water was commonly contaminated with the effluvia of pigsties and privies (Harker, 1988; Jennings, 1967). Additionally, drainage ditches overflowed in the rain, fouling wells (Jennings, 1967). From the journal of John Dickinson, a man living in Fewston parish, it can be seen that even by the late 19th century, water quality and sanitation had not improved. In 1881 Dickinson wrote

> 'There is talk about the deficient water supply and drainage of the village, and it is thought that the sanitary authority will take the matter in hand and compel the proprietors...to alter things. But the proprietors fear the cost and so we drink water highly polluted with sewage and our sinks are simply beastly. So much for the intelligent and industrious population who would suffer fevers and murrains sooner than spend a few pounds on purifying the water and improving the drainage' (Harker, 1988:48).

In 1884, the sanitation authority finally stepped in to start rectifying the problems (*ibid.*).

The presence of livestock in rural communities of North Yorkshire further affected sanitation and water. Depending on the location of farms, effluvia of livestock may have drained into waterways, thus fouling the water. Sheep washing was undertaken in streams and rivers, often in locations where humans would also

bathe (Alred, 1997; Harker, 1988). Given that animals were often housed under the same roof as their owners up to the 17th or 18th century (see Section 2.3.1.B) (Hoskins, 1953), their excrement were often only separated from the living areas of humans by a narrow passageway or screen, not necessarily enough of a separation for good sanitary conditions (Hey, 1986).

2.3.3 Pollution

'A dozen glass house cones, daily vomiting forth upon us a dense volume of smoke that hung as a pall over the locality, and obscured the windows and blackened the houses and roads.' John Imrie, South Shields
(Thornborrow, 1968:22-23)

A. Urban

One of the most commonly remarked upon features of Tyneside in the post-medieval period is the abundance of smoke produced through burning coal (Figure 2.5). Visitors to the area as early as the 17th century commented on the smoky atmosphere (Ellis, 2001b; Green, 2010; Hodgson, 1903; Hood Coulthard, 1959; Thornborrow, 1971a, 1988; Walton, 2000). For example, descriptions of South Shields recorded that there was 'such a cloud of smoke as amongst these [salt] works you cannot see to walk' and that the 'salt trade causes such a smoke that we could think the town were on fire' (Thornborrow, 1988:24). In the 18th century, Daniel Defoe could see the coal smoke from sixteen miles away while Lord Harley stated the houses there were 'in a perpetual thick, nasty smoke' (Thornborrow, 1988:24), and Celia Fiennes commented that the region 'is full of the Coale, ye sulphur of it taints ye aire and it smells strongly' (Fiennes, 1888:175). Continuing on into the 19th century, descriptions were provided of the 'turbid atmosphere' (Moffat and Rosie, 2005:230), the blackened buildings (Reid, 1845b; Thornborrow, 1968), and the 'copious diffusion of soot and coal dust which we daily breathe' (Robinson, 1847:12).

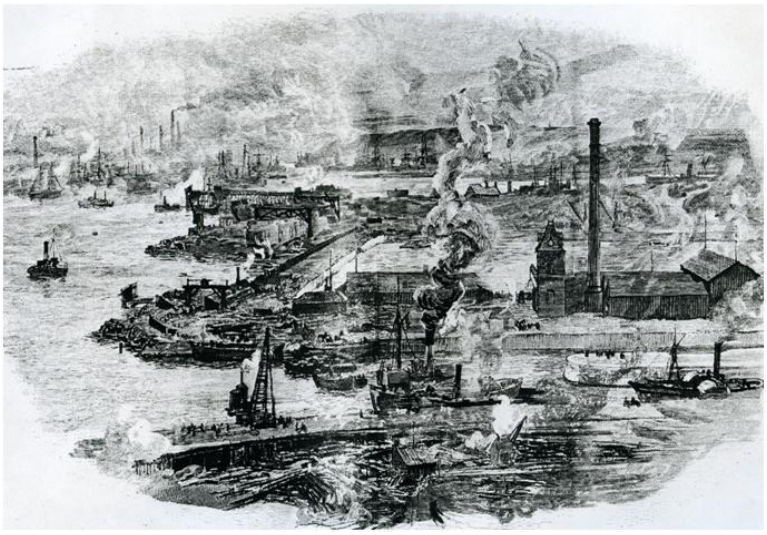

Figure 2.5: *General View of Albert Edward Dock* (North Shields) by Robert Jobling and William Lionel Wyllie from 1884 (Faulkner, 2010). This print demonstrates the smoky environment of North Shields with smoke produced from industrial chimneys and ships on the River Tyne.

The largest contributors to the smoke problem were the salt and glass industries. At the end of the 15th century, the salt industry changed their fuel source from wood to coal (Thornborrow, 1988). However, the corrosive sulphur dioxide released from the burning of coal destroyed the lead pans used to extract the salt. The adoption of iron pans solved this problem and also allowed for much larger pans to be created, leading to a higher demand for coal. It took around two and a half tons of coal to produce one ton of salt (Hodgson, 1903; Thornborrow, 1971a). This need for huge amounts of coal created a symbiotic relationship with the coal industry. "Small coal" that was unsuitable for domestic purposes due to its small size was instead bought by the salt industry. This coal burned inefficiently, producing copious amounts of smoke (Hodgson, 1903; Thornborrow, 1988). In 1643 there were 157 salt pans in use, later reaching a high of around 200 pans in the 18th century before declining in the 19th century (Levine and Wrightson, 1991; Thornborrow, 1971a, 1988).

As the salt industry was deteriorating, the glass industry was maintaining its level of production. Having started on the Tyne in the 18th century, the 19th century saw around twelve glass works in and around North Shields with a further twelve in South Shields (Linsley, 1992; Thornborrow, 1968). Similar to the salt industry, glass making was dependent on a ready supply of coal, with the tall glass furnace

chimneys continuously releasing smoke (Figure 2.6). In South Shields, an area near the location of several glass works formerly known as “Paradise”, because of its wooded area, had become a ‘blackened, smoking and sulphurous area’ (Thornborrow, 1968:22) due to the smoke pollution (Reid, 1845b). Prosecutions were regularly made against the glass industry over the discharge of smoke (Hodgson, 1903; Thornborrow, 1968). These prosecutions led to numerous temporary closures while the firms spent large sums of money attempting to remedy the situation. The introduction of a blast furnace, a perforated tower, and the substitution of coke for coal all lessened the amount of smoke released but ruined the glass, so these alterations were abandoned (*ibid.*). The salt and glass industries were certainly not the only sources of smoke in North and South Shields. Steam powered boats on the river, pit-engines at coal pits, and chemical works all contributed to the poor air quality (Hodgson, 1903; Hood Coulthard, 1960; Moffat and Rosie, 2005; Reid, 1845a, b). Additionally, westerly winds would blow the smoke from Newcastle-upon-Tyne, located further inland, to these more coastal towns. Despite the clear danger to health and vegetation caused by smoke, the general public was strongly in favour of the industries of the towns because so many of them depended upon them for employment (Reid, 1845b; Stanley and Derby, 1862).

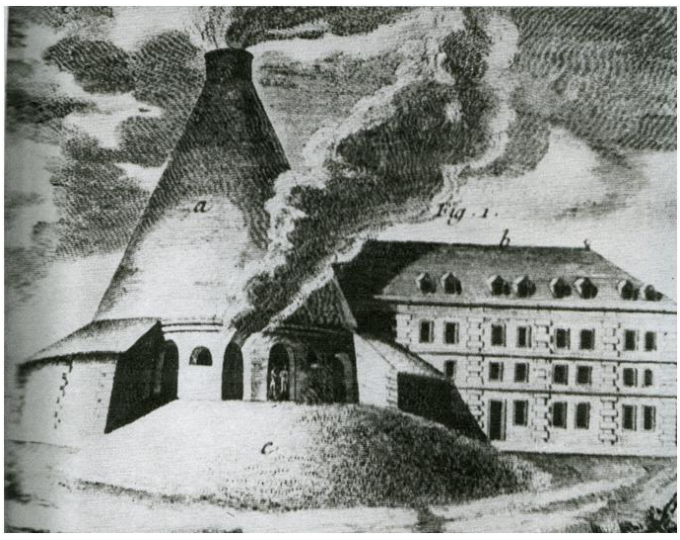

Figure 2.6: Print of a typical English glass house from Diderot’s *Encyclopaedia* in the 18th century (Buchanan, 1972).

Smoke was not the only by-product of the industries found in North and South Shields. The salt and glass industries produced a large amount of rubbish that was mixed with ballast from incoming ships and heaped on hills around South Shields, as it was forbidden to dump the rubbish into the river (Hodgson, 1903; Hood Coulthard, 1960; Thornborrow, 1968, 1988). As was stated in Section 2.3.1.A, lower class houses and other buildings were routinely built upon these rubbish heaps (Hodgson, 1903). As these hills consisted of cinders, incompletely burnt coals, ashes, and waste salt and lime, fires spontaneously started throughout the 18th and 19th centuries. These fires posed threats to human life not just through the fires themselves but also via the fumes emanating from the conflagrations (Hodgson, 1903; Stanley and Derby, 1862; Sykes, 1866a; Thornborrow, 1988). In one instance, ten men were nearly suffocated by sulphuric fumes while attempting to put out the fire (Sykes, 1866a).

Chemical enterprises, described as 'the monster nuisance of all' (Stanley and Derby, 1862:99) and 'noisome, hugely polluting, dangerous to health, but enormously profitable' (Moffat and Rosie, 2005:220-221), first appear at South Shields in 1756 (Thornborrow, 1971a). At least one alum boiling house at South Shields existed by this time (Pickles, 2002). In the first half of the 19th century, Tyneside was the most important centre of chemical manufacturing in Britain (Buchanan, 1972). By the 1840s, at least two alkali works and a vitriol (sulphuric acid) factory were located in the town with two additional alkali chemical works located just outside the town centre (Figure 2.7) (Reid, 1845b; Thornborrow, 1968; Warren, 1980). Around the same time, at least one alkali works was also located in North Shields (Warren, 1980). These chemical works produced bleaching powder, caustic soda, soda crystals (washing soda), bicarbonate of soda, refined alkali, and soda ash. Many of these products were used in the glass, soap, textiles, and paper industries (Atkinson, 1989; Dingle, 1982; Korzeniewicz, 1985; Warren, 1980). These products were created through combining sulphuric acid and salt to form saltcake and hydrochloric acid (Atkinson, 1989). The saltcake was then heated using huge quantities of coal and combined with limestone to make alkali (soda). Various other

materials were added to stages of the process to create the numerous byproducts sold onto other industries (Warren, 1980).

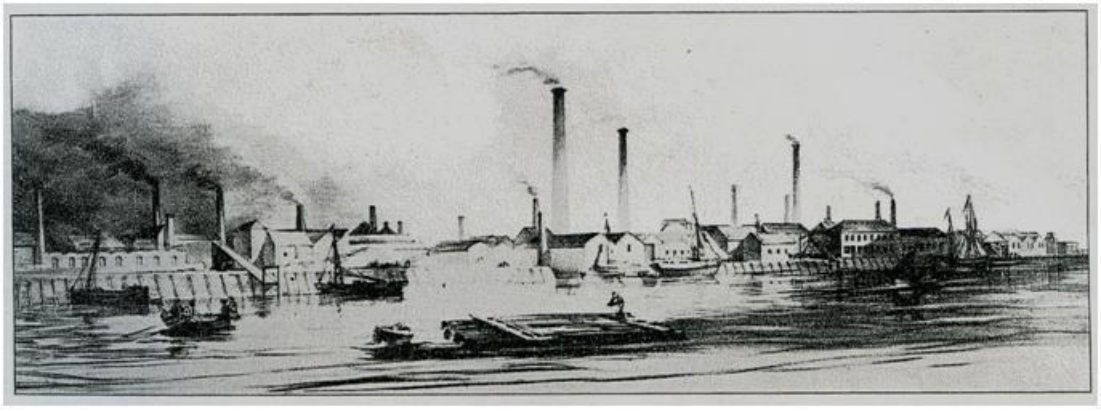

Figure 2.7: Jarrow Alkali Chemical Works at South Shields from about the 1840s showing the large numbers of chimneys and smoke associated with this type of industry (Hodgson, 1903).

The most common, but also the most wasteful, method of producing alkali was known as the Leblanc method, and was introduced to Tyneside around 1802 (Moffat and Rosie, 2005; Warren, 1980). This method led to large amounts of calcium sulphide dumped on refuse piles and hydrogen chloride and carbon dioxide released into the atmosphere (Campbell, 1968; Dingle, 1982; Moffat and Rosie, 2005; Warren, 1980). The gases released during the manufacturing process were painfully obvious to the general public. Owners of the factories at first tried to convince people that the gases were harmless and perhaps even beneficial to health despite the fact that they were damaging to vegetation; at one point it was even suggested that the gases could cure consumption (tuberculosis) (Atkinson, 1989; Dingle, 1982; Reid, 1845b). These tales did nothing to stop the complaints made against the industry. Chemical works were closed, sometimes permanently, because of prosecutions and complaints brought against them (Hodgson, 1903; Moffat and Rosie, 2005; Stanley and Derby, 1862; Thornborrow, 1971a; Warren, 1980). They were accused of issuing 'highly pernicious effluvia...which in some cases has nearly caused suffocation...not to mention the destructive influence of this insupportable nuisance on the adjacent pastures and gardens, where the herbage, fruit-trees, shrubs, and plants have withered, and the grounds in consequence become quite sterile' (Hodgson, 1903:367). Manufacturers claimed

they had spared no expense to limit the pollution produced by the industry, but that smoke and fumes were an inevitable part of industrial processes (Hodgson, 1903; Stanley and Derby, 1862). It was not until 1863 that an Act was passed requiring the use of towers to absorb much of the hydrochloric gas. This was more than 100 years after the alkali industry was first brought to Tyneside (Atkinson, 1989; Dingle, 1982; Warren, 1980).

B. Rural

Being removed from the centres of heavy industry did not protect rural Yorkshire communities from exposure to air and water pollution. Mining (coal and lead), milling grain, and the textile industry all had an effect on the surrounding land, water, and air with which people of North Yorkshire would have been in contact. Both corn and textile mills could be a source of large amounts of coal smoke (Figure 2.8). These mills were generally powered by a water wheel in streams but when the water levels got too low to turn the wheel, the mill turned to steam power. Craggs Mill in Norwood, Fewston parish, would use one ton of coal per day when relying on steam power (Alred, 1997). Other procedures within the textile industry were also responsible for water pollution. Dyeing and scouring would release water containing dyes, oils, tallow, and soap (Singleton, 1970). Retting (soaking hemp or flax in water to separate the fibres) was forbidden in streams by the Honour Court at Knaresborough due to the danger of causing harm to the water system and livestock (Jennings, 1967). There was also concern over the foul smell occurring during the retting process, leading to regulations against hemp being dried in ovens or over fires (Alred, 1997; Jennings, 1967). While large-scale production in the industry occurred in specialised buildings, small-scale production of most stages of textile manufacture occurred in the home, perhaps by all the members of the family, leading the waste products to be closer to living spaces than may otherwise have happened (Singleton, 1970).

Figure 2.8: West House Mill, Blubberhouses, Fewston parish (Alred, 1997).

Coal and lead were mined in various locations throughout Yorkshire. Since the 17th century, coke, a reasonably pure form of carbon, had been made in the coalfields (Atkinson, 1989). To make coke, the coal was heated in the absence of air to drive off the volatile chemicals as a gas (methane, carbon dioxide, carbon monoxide, benzene, coal tar, ammonia). These gases were vented into the atmosphere until the late 19th century. Lead ore dressing and smelting would also release lead and sulphur dioxide, both poisonous, into the atmosphere (Atkinson 1989). Flues were used in an effort to minimise the harmful effects, as some of the lead would deposit itself along the walls. This removed it from the fumes being released and allowed for collection of more lead (Atkinson, 1989; Jennings, 1967). These flues of course could not remove all of the poisonous toxins from the gases. A survey of 1813 in the area of a former iron ore mill described a 'naked hill' where the land was 'very much injured...not a blade of grass grew on the hill for a considerable distance around, the trees were blasted and withered' (Jennings, 1967:295). There were some communities less affected by the harmful nature of mining as they forbade industry from polluting their surroundings, although this was not a widespread practice (Hey, 1986). These hazardous conditions and changes to the environment have been shown through archaeological evidence. In North and West Yorkshire, peaks in the levels of lead, copper, and zinc from river sediments are associated with times of metal mining (Mighall *et al.*, 2004). Similarly, the increase in lead concentrations was associated with a decrease in

arboreal pollen, implying a loss of vegetation due to the atmospheric lead pollution caused by mining (*ibid.*).

2.3.4 Occupation

'In Shields the difference between the way a shipwright and a labourer could afford to live stood out sharply - in fact, labourers' families had little chance of ever rising above the poverty line.'
(Foster, 1975)

A. Urban

Nearly every commercial enterprise in North and South Shields in the post-medieval period was connected to the coal industry. In the 19th century about half of the labour force of South Shields was involved in the movement of coal from County Durham: 25% of the work force was seamen/sailors, 10% shipbuilders, and 10% keelmen moving coal from the shore to ships (Foster, 1975; Levine and Wrightson, 1991; Reid, 1845b). Pitmen were responsible for the extraction of the coal before passing it on to keelmen to transport the coal to the ships (Atkinson, 1989; Simpson, 1988). From there it was the sailors', master mariners', and ship-owners' responsibility to deliver the coal to trading ports along the east coast of Britain and further afield (Charlton, 2008; Simpson, 1988). To keep the trade of coal running, shipbuilders, block and mast makers, rope makers, sail makers, chain makers, and anchor smiths were also needed (Hodgson, 1903; Hood Coulthard, 1960; Phillips, 1894; Sykes, 1866a). As the coal industry wanted to dispose of small coals that were not suitable for sale on the domestic market, and ballast was often in the form of sand brought back on return journeys of ships, this led to the formation of salt, glass, and chemical industries (Atkinson, 1989; Campbell, 1968; Ellis, 2001b; Hodgson, 1903; Thornborrow, 1968, 1988). At the peak of industry, 75,000 tons of salt was produced in seven years at South Shields alone (Hodgson, 1903). Additionally, South Shields produced one sixth of the total glass in the United Kingdom, with the 10,000 men employed in the alkali industry on Tyneside producing more than 50% of the national output (Campbell, 1968; Lendrum, 2001; Thornborrow, 1968). These industries were strong until a decline came in the 19th

century due to rising costs and competition (Hodgson, 1903; Hood Coulthard, 1960; Thornborrow, 1971a, 1988). The retail aspects of the communities (*e.g.* butcher's shops, shoemaker's shops, and inns/public houses) were present as much for the general population as for the shipping industry (Hood Coulthard, 1960). Other information specific to either North or South Shields is discussed below.

Industry in North Shields has been focused on its port since at least the medieval period. Coal, salt, and hides were exported while fish, wool, and wine were imported (Simpson, 1988). The fish brought in from ships was smoked and salted in the town. Evidence for skinning, glove making, and anchor smithing is seen from the 17th century (Phillips, 1894). From at least the 18th century commercial ventures have included tanneries, roperies, lime works, shipbuilding, and timber yards (Lovell, 1991; Reid, 1845a; Simpson, 1988; Sykes, 1866a). The 17th century also saw North Shields become one of the leading producers of salt in the country, as discussed above (Thornborrow, 1988). In 1800 there were about twelve glass works in and around the town but it is unknown when this industry first arrived in the town (Linsley, 1992; Thornborrow, 1968).

Looking at the Quaker population of North Shields, the few occupations known for them show they were largely engaged in skilled and mercantile businesses. For example, the Richardson Tannery, opened in the 18th century, and a ropery were owned by Quaker families (Lovell, 1991; Simpson, 1988). Occupations listed for fathers of children being baptised between 1675 and 1839 can be found in Table 2.2. Other occupations known for Quakers in North Shields included supervisors of the excise and anchor smiths (Hughes, 1952; Phillips, 1894).

Occupation	No. of Individuals
block and mast maker	1
broker	1
cordwainer	1
draper	5
grocer and tea dealer	3
hatter and hosier	1
insurance and ship broker	1
labourer	1
mariner	3
master mariner	4
mercer	1
miller	2
painter	1
rope maker	1
shipmaster	2
shopkeeper and auctioneer	1
skinner and glover	1
tanner	2
timber merchant	1

Table 2.2: The occupation of fathers listed in baptism records in the Newcastle Monthly Meeting (Quaker) for individuals from North Shields (MF176).

Much like North Shields, South Shields developed its industries in the medieval period based on its geographical location. At that time, coal, salt, and shipping industries were founded (Openshaw, 1978). The salt industry was first established in the town before the end of the 15th century and the 17th century saw South Shields become the largest salt producer in England (Thornborrow, 1971a, 1988). By the 18th century, the development of glass production in the town had established South Shields as an important industrial town (Hodgson, 1903; Openshaw, 1978; Thornborrow, 1968, 1971a). At least three glass houses were founded in the 18th century and were still in production well into the 19th century (Ellison, 1975). In 1832, eight large glass factories functioned in the town, making it the largest glass manufacturer in England but, by 1886, only one glass works remained (Hodgson, 1903; Hood Coulthard, 1960; Thornborrow, 1968, 1971a). The ceramic industry started developing in 1721 (Openshaw, 1978) with the first chemical manufactory at South Shields appearing in 1756 and surviving into the 20th century (Thornborrow, 1971a). In 1781 at least ten ship repair yards were also

opened (Openshaw, 1978). Throughout this time, coal mining became increasingly important. The first reference to a pit in the town was made in 1789 and pits were still being opened in the 19th century (Hood Coulthard, 1960; Thornborrow, 1971a). By 1845, South Shields was considered part sea-port, part manufacturing town, part mining population (Reid, 1845b). The multipurpose nature of the settlement is demonstrated by two occupation surveys and parish registers (Table 2.3). A shipping directory of 1850 revealed eight boat builders, fourteen ship builders, five chain/cable makers, 142 master mariners, 123 ship owners, fifty boot/shoe makers, nine brewers, 174 inns/hotels/public house keepers, three salt manufacturers, one varnish manufacturer, one violin string maker, one professor of music, and six straw bonnet makers (Hood Coulthard, 1960). In 1873 a survey of occupations showed that with a population of about 36,500, South Shields had 91 butchers, fifteen bakers, 145 inn keepers, 102 beer retailers, four doctors, and eight registered pharmacists (Campbell, 1975). Additional industries noted at South Shields were soap manufacturing, a tilery, a brickworks, an ironworks, timber yards, a ropery, and lime kilns (Hodgson, 1903; Reid, 1845b).

Employment in the urban centres of North and South Shields could be uncertain and low paying. Work was often seasonal and centred on the ups and downs of the coal and shipping industries, particularly in the 19th century (Ellis, 2001b; Woodward, 1995). In many industrial towns, income from wives and children could bolster the family income, but the industries at North and South Shields offered few employment opportunities for women and children, forcing families to rely on the older males to support the household (Foster, 1975; Woodward, 1995). Throughout the post-medieval period, many tradesmen did engage in various by-employments or work outside of their “normal” occupation in an effort to improve the household’s earnings (Atkinson, 1989; Woodward, 1995). This may have helped keep families afloat in difficult economic situations.

Occupation	1797/98	1827	Occupation	1797/98	1827
attorney/solicitor	1	1	mariner	115	203
baker	1	2	mason	2	12
ballast conveyor	2	3	master mariner	10	17
blacksmith/anchor smith	2	11	merchant	3	3
			painter & glazier	5	3
block & mast maker	2	3	pilot	10	13
brewer	1	2	pipe maker	-	1
brick maker/layer	3	2	pitman	-	38
butcher	3	4	publican	-	1
carpenter/joiner	16	12	roper/rope maker	6	6
chemist & druggist	-	3	saddler	-	1
clerk/office work	4	3	sail maker	1	5
clock & watch maker	-	1	sheriff's officer	-	1
coach man/footman	1	1	ship owner	3	1
cooper	-	5	ship/boat builder	59	81
drayman/cart man	2	4	shoemaker/ cordwainer	11	25
engineer	-	4			
farmer	4	5	soldier	3	-
gentleman	1	-	surgeon	1	1
glass industry	21	20	tailor	5	6
grocer	-	3	teacher	-	1
hair dresser	1	2	textiles	4	1
hatter	-	1	tile maker	-	2
husbandman	9	9	trimmer	8	5
iron founder/monger	-	4	waterman	1	-
keelman/wherryman	14	9	yeoman	-	2
labourer	10	20	**Total**	345	563

Table 2.3: The occupation of fathers listed in baptism records at St Hilda's Church, South Shields (MF831).

As early as the 17th century, the local industries proved to be unstable. In 1655 the Council of State was petitioned, arguing that 1,000 salt workers would be out of work if free trade with Scotland commenced (Howell, 1967). The case was defeated and the decline of the salt industry on Tyneside began. The year 1815 saw a sudden reduction of the navy due to economic depression, leading to a large number of seamen being suddenly unemployed in the port towns of Britain. Great numbers of these sailors accumulated at North and South Shields, leading to protests (McCord, 1970; Sykes, 1866b; Thornborrow, 1971b). In addition to the seamen being put out of work, shipwrights and other shore workers found

themselves unemployed due to the same economic difficulties (Hodgson, 1903). These were not the only protests seen in North or South Shields in the 18th and 19th centuries as keelmen, shipwrights, and seamen all protested their low wages (Foster, 1975; Hodgson, 1903; Hood Coulthard, 1960; McCord, 1970, 1971). Sailors were brought in to replace the striking workers but generally talks broke down without much ever being achieved. These strikes were mostly peaceful but one man in North Shields was shot and killed by marines during a protest in 1819 (Moffat and Rosie, 2005).

B. Rural

The industrial revolution went hand in hand with the agricultural revolution. Farmers were encouraged to maximise their output through enclosure of commons and mechanisation in order to provide for the wage-labourers amassing in the urban centres (Drummond and Wilbraham, 1957; Gidney, 2009; Green, 2003). Agriculture was the principal occupation for those living outside of major towns from the late 1600s up into the 19th century, and the people of North Yorkshire were no exception (Hey, 1986; Singleton, 1970). Even after that time, agriculture was still practised by the majority of rural Yorkshire dwellers even if it was not their main form of employment (Hey, 1986; Jennings, 1967; Woodward, 1995). At Wharram Percy, sheep were reared for their milk, meat, wool, and manure (Richardson, 2010). In North Yorkshire more broadly, miners and those in the home textile industry were almost always subsistence farmers in addition to their waged employment owning or renting small pieces of land. Once textile work moved from the home to mills, this side practice of farming greatly diminished because of the long hours spent at the mill (Hey, 1986).

Major industries in North Yorkshire included textiles and mining (coal, iron, or lead, depending on the area). Men, women, and children were all employed in both of these industries, and the majority of workers in the textile mills were women and children (Alred, 1997; Hey, 1986; Jennings, 1967). The commercial textile industry started in the 17th century, with home spinning as a supplement to farming (Alred, 1997; Clark, 1999; Jennings, 1967). Merchants would bring the raw

wool, flax, or hemp to the spinners and then collect the spun material to deliver it to weaving shops where several people would work together. Textile mills were eventually built starting in the late 18th century, sometimes on the site of corn mills. The first one in Fewston parish was built in 1791 (Alred, 1997; Jennings, 1967). At the height of textile production in Fewston in the early 19th century there were four mills in operation, spinning cotton, flax, or hemp, depending on demand (Alred, 1997; Jennings, 1967). Mining in North Yorkshire dates to at least the early 16th century when there was iron mining and smelting taking place in the parish of Fewston (Jennings, 1967). Lead mining was present from the 16th century, and coal mining started in the region in the 17th century (*ibid.*). Lead smelting took place at seven smelting mills in the region using a combination of water power and peat fires in the 19th century (*ibid.*). It should be noted, that much of this industry was commonly located outside of the domestic areas in rural regions as small population densities produced fewer demands on space. These industries being removed from settlements may have lessened the exposure of individuals residing in the parish.

Rural communities and small market towns of North Yorkshire had small-scale industries based on craftsmen who were seldom able to specialise due to lack of demand, but instead they were "jacks of all trades" (Hey, 1986; Woodward, 1995). For example, John Dickinson of Fewston Parish, listed his occupations in one year as mason, farmer, pork salter, paraffin salesman, teaseller, manure company agent, hay trader, insurance agent, way warden, planning inspector, overseer, census official, rate collector, vaccination officer, parish councilor, registrar of births and deaths, trustee, and secretary and grand master of a Friendly Society (Harker, 1988). Three corn mills were located in Fewston Parish, the earliest dating from the 16th century, and they were largely for milling and drying oats (Alred, 1997; Jennings, 1967). Seventeenth century records reveal several occupations including roper, yeoman, and husbandman (Neave and Neave, 2006). A brewer and a blacksmith were recorded from 1699 (Alred, 1997). Official Returns (census data) from 1821 for Fewston Parish listed twelve gentlemen, twelve farmers, four victuallers/publicans, five blacksmiths, three flax and tow spinners, two linen

manufacturers, two stone masons, three grocers, three shoemakers, and one each of a flax dresser, miller, tanner, cabinet maker, wheelwright, tailor, steward, and vicar (Baines, 1822). Similar occupations were seen in the parish registers for Fewston (Table 2.4), but there is an interesting trend shown. While farming was a common occupation through the period, employment in the textile industry increased in the 19th century.

Only one occupation was given in the 1821 Official Returns for Wharram Percy Parish: farmer (Baines, 1823). A more extensive list of other occupations was recorded in the parish registers and can be found in Table 2.5.

Poverty may not have been common in North Yorkshire in the 19th century (Hey, 1986). There were periods of hardship due to particularly bad harvest failures or trade depression but, in general, able-bodied men and women would have been able to find employment even if it was seasonal or part-time (*ibid.*). However, cottagers would have been particularly hard hit by economic declines as they were not self-sufficient, instead relying on wage-labour (Jennings, 1967). In areas of Northeast England at times during this period, wages paid to agricultural workers were higher than average, probably due to the availability of well-paid work in nearby industrial centres (Atkinson, 1989; Drummond and Wilbraham, 1957; Hey, 1986). The improvements made to agriculture such as mechanisation, leading to unemployment and loss of land, meant that many farm workers protested or rioted in other areas of the country. However this was not seen in North Yorkshire as these farm hands were generally not unemployed for long (Atkinson, 1989), with readily available work in the mines and industries located nearby.

Occupation	1738	1742	1743	1745	1813	1825	1834	1842	1857	1866
blacksmith	1	1	1	1	-	2	1	-	1	1
butcher	-	1	-	2	-	-	1	-	1	-
carpenter/joiner	-	-	1	-	1	2	1	4	-	2
clerk	-	-	-	-	1	-	-	-	-	-
common carrier	-	-	-	-	-	1	1	-	-	-
cooper	-	-	-	-	-	-	-	1	-	-
farmer	2	5	10	4	25	23	23	27	10	16
gentleman	-	-	-	-	-	-	-	-	-	1
hawker	-	-	-	-	-	-	1	-	-	-
householder	-	-	-	1	-	-	-	-	-	-
husbandman	-	1	1	-	5	3	-	-	-	-
innkeeper	-	-	-	-	1	1	3	-	-	-
labourer	1	-	3	7	13	8	7	6	3	-
lodging house keeper	-	-	-	-	-	-	-	-	-	1
manufacturer	-	-	-	-	-	1	1	-	-	-
mason	-	1	2	-	1	-	1	1	-	2
mechanic	-	-	-	-	-	-	1	-	-	-
miller	1	1	-	-	1	1	-	1	1	1
panner[?]	-	-	-	-	-	-	1	-	-	-
quarry man	-	-	-	-	-	-	-	-	-	1
road maker	-	-	-	-	-	1	-	-	-	-
saddletree maker/ saddler	1	-	-	-	-	-	-	1	-	-
school master	-	-	-	-	-	1	-	1	-	-
shoemaker/ cordwainer	2	-	-	2	3	5	4	1	3	-
shop keeper	-	-	-	-	-	1	-	-	-	-
tailor	-	-	-	-	1	1	2	2	2	-
tanner	-	1	1	-	-	-	-	-	-	-
textiles	-	2	1	1	11	13	20	16	-	2
vicar	-	-	-	-	-	-	-	-	1	-
waggoner	-	-	-	-	-	1	1	-	1	-
whitesmith	-	-	-	-	1	-	-	-	-	-
yeoman	3	-	-	-	-	5	3	2	-	-
Total	11	13	20	18	64	70	72	63	23	27

Table 2.4: The occupation of fathers listed in baptism records at the church of St Michael and St Lawrence, Fewston (Parkinson, 1899; PR/FEW 1/16, 1/17).

Occupation	1810s	1820s	1830s	1840s	1850s	1860s	1870s	1880s
blacksmith	1	-	-	-	-	4	1	-
brick maker	-	-	-	1	-	-	-	-
bricklayer	-	-	-	-	-	1	-	-
butcher	-	-	-	-	-	-	1	-
carpenter	-	1	3	-	-	-	-	-
carrier	-	-	-	2	5	4	-	-
farm bailiff	-	-	-	-	1	-	-	-
farm servant	-	-	-	-	-	1	-	-
farmer	19	33	17	11	12	9	7	2
game keeper	-	-	-	2	1	-	1	2
hind	-	-	-	-	-	4	1	2
inn keeper	-	-	-	1	-	1	-	-
joiner	-	-	-	-	-	1	-	-
labourer	27	41	50	52	52	58	12	10
miner	-	-	-	5	10	1	-	-
mining engineer	-	-	-	-	-	-	-	1
parish clerk	-	-	-	1	-	-	-	-
plate layer	-	-	-	-	1	-	-	-
publican	-	-	1	-	1	3	-	-
school master	-	-	-	1	-	-	-	-
servant	1	4	4	-	-	2	-	-
shepherd	3	1	1	-	2	-	-	5
shoemaker	-	-	6	2	-	1	1	-
station manager	-	-	-	-	-	-	-	1
stone mason	-	-	-	1	-	-	-	-
tailor	1	-	-	2	-	3	-	-
wheel wright	-	-	-	2	-	-	-	-
yeoman	1	-	-	-	-	-	-	-
Total	53	80	82	83	85	93	24	23

Table 2.5: The occupation of fathers listed in baptism records at St Martin's church, Wharram Percy (PR/WP 3).

2.3.5 Diet

'I do not believe that any Englishman who is his own master has ever eaten a dinner without meat.' P. Kalm, 1748
(Wilson, 2003:98)

The diets of people living in northern urban or rural environments were not that dissimilar from each other in the post-medieval period (Drummond and

Wilbraham, 1957). Food prices in the north of the country were generally lower than those in the south (Hey, 1986) and even after bad harvests, people in the north were still able to access a wide variety of foodstuffs, regardless of socioeconomic status (Drummond and Wilbraham, 1957). The labouring poor's diet consisted of oatmeal, tea, milk, butter, potatoes, bread, and meat when it could be afforded (Drummond and Wilbraham, 1957; Hey, 1986). This was reduced to bread, butter, and tea or water in lean times (Ketabgian, 2007; Wilson, 2003). Tea, coffee, and chocolate were introduced to Britain in the middle of the 17th century, with the price of tea dropping far enough that it was becoming a general drink in the 18th century (Drummond and Wilbraham, 1957; Freeman, 1989; Gray, 2009; Ketabgian, 2007; Wilson, 2003). Due to its connection to tea, sugar spread across the social classes, beginning in the 18th century, and had reached even the poorest by the 1820s (Charlton, 2008; Drummond and Wilbraham, 1957; Wilson, 2003).

A division could be seen between the classes unrelated to financial state since there was a common belief that the labouring poor could tolerate heartier foods than the wealthier classes (Thirsk, 2006). For example, hard cheeses and salted fish were too coarse for the upper class but perfectly acceptable for the poor. Beliefs such as this dictated eating habits, perhaps to the detriment of the upper classes. Foods deemed unsuitable for the wealthy included most dairy products, vegetables, and fruit (Freeman, 1989; Giorgi, 1997; Thirsk, 2006). The late 17th century saw some shifts in these beliefs, with the wealthy discovering dairy, in particular cheese and butter (Drummond and Wilbraham, 1957; Thirsk, 2006; Wilson, 2003). This growing demand for cheese and butter meant it was more profitable to make these goods than to sell milk, leading to milk shortages amongst the poor of the north. Various vegetables also gained acceptance as continental food habits were adopted (Thirsk, 2006). Fruit and vegetables that were imported or rare were popular amongst the rich but lost their appeal as soon as they were deemed common, and were only from then on acceptable for the poor (*ibid.*). Meat was also divisible by class. Certain cuts and types of meat were favoured by the middle and upper classes, keeping the prices high, but this did leave less popular cuts and offal at lower prices that could be afforded by the lower classes (*ibid.*).

A. Urban

Where the diets of these two settlement types differed was the level of freshness/quality of the food and how the foodstuffs were acquired. With the rise of urbanisation, there seemed to have been a deterioration in the quality of foodstuffs available in towns, particularly in those likely to be bought by the poorer classes (Drummond and Wilbraham, 1957). Dairy farmers in Yorkshire sold their best quality food to London and sent their poorer grade produce to the northern industrial towns (Hey, 1986). Milk sold in towns was generally of very poor quality and was a frequent source of infections (Drummond and Wilbraham, 1957). Milk was also delivered door to door in open pails which of course could have potentially been contaminated (Hood Coulthard, 1959; Wilson, 2003).

Adulteration of foodstuffs was also seen in urban centres although no specific reference to this occurring in North or South Shields has been found. Meat and milk sold as fresh were likely to have been in a "dubious state" (Drummond and Wilbraham, 1957; Freeman, 1989). Tea was mixed with dust, floor sweepings, sand, or cheap herbs while beverages such as milk, beer, and wine were watered down with water that may have been polluted (Drummond and Wilbraham, 1957; Freeman, 1989; Gray, 2009; Ketabgian, 2007; Wilson, 2003). Lead was also added to wine in an effort to sweeten it, and flour and chalk were added to milk to whiten it. As late as 1931, 10% of milk samples from Newcastle-upon-Tyne had been adulterated (Drummond and Wilbraham, 1957). Alum was certainly added to bread, and other possible additions included chalk, lime, bone, and white lead in an effort to whiten it (Drummond and Wilbraham, 1957; Freeman, 1989; Wilson, 2003). Most vinegar on the market was likely to have been vitriolic acid (sulphuric acid) or oil of vitriol (Drummond and Wilbraham, 1957). Meanwhile, copper salts were added to make vegetables appear greener. As for acquiring foodstuffs, many people in rural North Yorkshire were either self-sufficient or grew some of their own produce. All of the foodstuffs consumed by urban individuals had to be purchased, most people would have been unable to grow their own vegetables or own livestock (Alderman, 1986). The consumption of vegetables by the working classes in towns was low compared to their rural counterparts, and this was due to their

inability to access them for free through gardening or finding wild growing options (Drummond and Wilbraham, 1957; Freeman, 1989; Thirsk, 2006).

However, there was some advantage for urban dwellers, because being in a port city helped protect against poor harvests and food shortages. Corn imported from abroad and other foodstuffs would have been available in towns but these goods seldom reached into the rural hinterland (Thirsk, 2006). Port towns such as North and South Shields had greater access to a wider variety of food stuffs due to imports, but also because food from the local region arrived to be either exported or sold in the town (Thirsk, 2006). For many workers in the northern industrial towns, choosing their meals relied upon finances. Grain/bread choice was generally made according to cost, even up until 1800 (Woodward, 1995). Bachelors and small families would have been able to occasionally afford beef or cheese. Other foods would have supplemented the diet when prices fluctuated low enough so that they would not have had to rely solely on grain products. Larger families would have often experienced a lack of food, with the children of working men suffering chronic malnutrition, but there were some success stories of families having a higher standard of living (Woodward, 1995).

Many butcher shops were present in both North and South Shields, and located there as much for the general population as to supply the vast numbers of ships with food rations (Hood Coulthard, 1960). As the urban populations grew, there was an increased demand for beef over butter and cheese, the former being a source of protein for the working poor (Gidney, 2009; Wilson, 2003). Use of communal or baker's ovens for baking or preparing hot meals was in place in towns from the medieval period and was often necessitated because of the expense of fuel and the lack of hearths or ovens in the home (Thirsk, 2006). At South Shields, public ovens were still in use in the 1870s (Drummond and Wilbraham, 1957; Hood Coulthard, 1959).

B. Rural

In North Yorkshire most individuals, if not self-sufficient, at least produced some of their own foodstuffs (Hey, 1986; Jennings, 1967; Woodward, 1995). Miners and individuals involved in a vast array of employment farmed or at least maintained a garden. Most families grew their own vegetables and potatoes, and often were able to produce their own dairy products (Jennings, 1967). Before the Enclosure Acts excluded access to communal fields, many were also able to keep sheep and other animals for meat and wool. Many would also have had the opportunity for collecting wild vegetables, fruits, and herbs or snaring wild rabbits and birds (Thirsk, 2006; Wilson, 2003). In addition, food and drink was sometimes included as wages paid to labourers (Drummond and Wilbraham, 1957; Freeman, 1989; Pennell, 1999; Woodward, 1995). This food was consumed at the worksite, meaning the worker was catered for, but not his family at home. This was a particular problem since wages were sometimes lower to include the cost of the food or drink.

In a typical household in North Yorkshire, the most common breakfast and supper dishes were oatmeal dumplings cooked in milk with coarse bread (Hey, 1986). Little fresh meat was eaten, instead relying on bacon and salted beef or homemade cheese for protein (Hey, 1986; Jennings, 1967). Oats or oatmeal was the staple of the diet in the region with homebrewed ale as the main drink, although tea started to replace it in the late 18th century (Jennings, 1967; Thirsk, 2006). At Wharram Percy, archaeological research has discovered that domesticated sheep, cattle, and pig provided the majority of the meat, with game, marine fish, and shellfish occasionally entering the diet (Richardson, 2010). Marine fish arrived from the coast either whole (and usually fresh), or dried (Barrett, 2010).

Oats were the predominant crop for North Yorkshire from at least the latter half of the 16th century, but rye, wheat, and barley were also grown. Turnips and rape were seen in some places as early as the 1660s but had spread more widely by 1750 (Jennings, 1967; Wilson, 2003). These were grown as field crops and sown in fields that would otherwise have been left fallow, thus increasing land productivity

and allowing more livestock to be wintered. This would have enabled greater access to fresh meat year-round. Potatoes were introduced into the area by 1713 but were more commonly seen after 1731 (Drummond and Wilbraham, 1957; Jennings, 1967). For a sample of the quantities of various crops that were grown in Fewston parish, a survey of 1801 can be examined. The 1,207 arable acres consisted of 899 acres of oats, 68 of wheat, 57 of barley, one of rye, 87 of turnips/rape, 77 of potatoes, and eight of peas/beans (Jennings, 1967).

Changes in technology inadvertently led to changes in diet. Durham shorthorn cattle were bred to produce a fatty meat much prized by miners as it was thought to give them more energy (Atkinson, 1989). The fat from the cattle was also used in the production of soap and candles. As gas was used more and more for lighting, the market for candles correspondingly decreased. Durham shorthorns then became more useful for dairy than for meat/fat production, leading to an increase in dairy production in North Yorkshire. The consumption of mutton decreased as the wool textile industry expanded, and sheep became more profitable as wool than as meat producers (O'Connor, 1989).

2.3.6 Health, Health Care, and Mortality

Disease 'is the offspring of filth, nastiness and confined air, in rooms crowded with many inhabitants…I think we may without much hesitation pronounce that the occasional cause of it is human effluvia which has been generated in some little dirty confined place, of which there are great numbers in…every…manufacturing town' John Haysham, 1782 (Chadwick, 1842:25)

A. Urban

From the 16th through to the 19th centuries there were serious outbreaks of infectious disease, particularly in the poorer areas of North and South Shields (Hodgson, 1903; Openshaw, 1978; Wrightson, 2009). Cholera, pulmonary tuberculosis, and smallpox were ubiquitous (Horsley, 1971) and plague was one of the earliest recorded diseases in the region. Outbreaks of plague on Tyneside occurred in 1544-1545, 1570-1571, 1576, 1579, 1588-1589, 1593, 1597, 1604, 1625, and 1636 (Levine and Wrightson, 1991; Wrightson, 2009). The 1635 plague was

particularly devastating, and it entered the region in October at North Shields, having come from Holland (Howell, 1967; Wrightson, 2009). The disease was largely dormant through the winter but its severity was obvious by May and continued to get worse. The plague nearly depopulated North Shields, and in Newcastle-upon-Tyne at least 5,600 individuals died, which was around 47% of the population (*ibid.*). Fevers were reported in 1790 and 1794, after which efforts were made to clean the streets and remove standing filth (Hodgson, 1903).

Cholera struck in the 19th century; having first arrived at Sunderland it spread rapidly, with epidemics occurring in 1831-1832, 1848-1849, 1853-1854, and 1866 (Atkinson, 1989; de Montluzin, 2002; Hodgson, 1903; Reid, 1845b; Moffat and Rosie, 2005; Thornborrow, 1971a). With the outbreak of cholera in 1831, a Board of Health consisting of clergy, medical men, and important individuals of South Shields was formed to prevent the spread of the epidemic which they attributed to bad air (Hodgson, 1903). They employed additional street sweepers and took measures to have all courts and closes cleaned and whitewashed. They also wanted to restrict the entry of vagrants into the town and increase the number of watchmen to keep these vagrants out. By the start of 1832, prayer was resorted to in a last effort to eradicate it, all businesses were shut and intercession services were held at St Hilda's and other chapels. Parish registers at St Hilda's, South Shields record twenty-nine deaths due to cholera out of seventy-one burials between October 1832 and October 1833 (PR/FEW 1/19). In the epidemic of 1848, 150 people died in South Shields while there were 44 deaths from cholera in just two months in 1853 (Hodgson, 1903; Thornborrow, 1971a). The epidemic of 1866 was met with the erection of a wooden cholera hospital, and the first case in this epidemic was a sailor on a ship that had recently arrived from Hamburg (Hodgson, 1903). At this time, there were no precautions in place, such as inspections of vessels from foreign ports to prevent the importation of disease. Between the outbreaks of cholera there were periods of generally better health in the region. In 1845, no epidemic disease or "contagious fever" had been reported for several years in North Shields and, aside from cholera in 1832, there had been no epidemics in South Shields since 1819 (Reid, 1845a, b).

Everyone living in North and South Shields would have been exposed to the pollution and refuse created by the industries located there, but the individuals actually employed in the manufacturing processes would have been even more greatly exposed than the average person, which would have potentially affected their health. Individuals employed in the glass and salt industries would have been exposed to copious quantities of coal smoke on a daily basis, as it took around two and a half tons of coal to produce one ton of salt (Thornborrow, 1971a), and it is considered that South Shields alone produced 10,000 tons of salt a year (Howell, 1967). This is certainly no small amount. Plumbers, meanwhile, were constantly exposed to lead as they cast or refined lead and its ashes (Woodward, 1995). The hydrochloric acid fumes and carbon dioxide produced from the alkali works were observed to be harmful to vegetation but, even by 1845, there was still no consensus as to whether these fumes were detrimental to human health (Reid, 1845b) but many workers in the alkali industry did die from industrial diseases (Warren, 1980). Both men and women were employed in the coal pits, which entailed working underground all day. As the mines lacked ventilation, this led miners 'to suffer the most awful agony in an exceedingly high temperature, inimical to his health, comfort, and even life' (Hodgson, 1903:374).

Mortality rates differed between urban and rural regions. In 1841, one in 150 individuals in Newcastle-upon-Tyne died from epidemic diseases compared to one in 334 in the more rural northern districts of Northumberland (Robinson, 1847). Mortality from respiratory diseases such as tuberculosis was twice as high in Newcastle compared to rural county districts (*ibid.*). Mortality also varied somewhat within urban areas. The overall mortality rate for North Shields was 2.98% with the highest mortality rates occurring in the older, more crowded and derelict areas of town (Reid, 1845a). Looking at three regions of the town, the mortality rate in the well-drained, better-off Bedford Street was 1.863%, with a 2.0% rate in the undrained Low Lights area and the highest rate (2.22%) in the partially undrained Stephenson Street (*ibid.*). More drastic differences were seen at South Shields. For the year 1843, the mortality rate was 2.24% but rates varied between areas of the town (Reid, 1845b). The lowest rate was 1.31% in a well

ventilated and drained area of town inhabited largely by glassmakers. The highest rate was 3.73% in one of the worst areas, where houses were built entirely on the ballast hills (see Section 2.3.1.A) and the region contained some of the filthiest conditions in the town. This area was inhabited by glassmakers, sailors, and workers in the alkali factories. In 1843 the average age at death was 26.02 years with nearly half of all deaths occurring in those under the age of five (Reid, 1845b). The causes of death for 1843 were consumption (88 individuals), fever (20), chest/respiratory diseases including pneumonia, bronchitis, asthma, and pleurisy (69), intestinal diseases (32), smallpox (32), measles/scarlatina/whooping-cough/croup (37), atrophy/debility in infancy (97), decay/natural decay or natural causes in old age (58), and other diseases and childbirth (201) (*ibid.*).

Medical care was available in various forms in North and South Shields. The medical professions were present and, in general, people of all classes sought out health care when it was needed (Reid, 1845a, b). In some cases the care was provided without charge, for example after a fever outbreak in 1794 the South Shields Vestry paid for a doctor to visit the poor sick (Hodgson, 1903; Reid, 1845a). By 1873 South Shields boasted four doctors and eight pharmacists, treating a population of about 36,500 (Campbell, 1975). A dispensary was established at North Shields in 1802 which had six medical practitioners by 1845 (Reid, 1845a; Sykes, 1866b). The dispensary averaged 620 patients, plus about double that in unregistered casualties (Reid, 1845a). The dispensary at South Shields annually admitted about 1,300 patients while 770 to 800 patients were seen by the parish surgeons (Reid, 1845b). Visiting a doctor or pharmacist was not always possible due to the financial costs involved (Campbell, 1975), while others were kept away due to fear of catching a fever in hospital or the thought of the excruciating operations (this was before anaesthesia). For instances such as this, less reputable medical advice was also available, with a survey of proprietary medicines for 1812 finding there were 550 on offer (Campbell, 1975). One of the most common proprietary medicines was the “cure-all,” reputed to cure any complaint the sufferer might have (*ibid.*). A second common type of proprietary medicine were those containing opium derivatives and used for all age groups, resulting in many deaths across

Britain. Itinerant quack doctors selling these "medicines" and services such as removal of warts were common features of the marketplace (Hood Coulthard, 1960).

B. Rural

There is little evidence for commonplace or endemic infectious disease in rural North Yorkshire in this period, although they did "visit" on occasion. The 1720s saw the last of a large numbers of deaths due to fevers following devastating harvests (Hey, 1986). In John Dickinson's diary from Fewston Parish he recounts several infectious diseases in the late 19th century (Harker, 1988). His young daughter caught measles at the age of about six but he does not report any other cases in the village. The schoolmaster passed away from typhoid fever, and because the villagers feared the spread of the disease they locked his body in his house for three days. However, this did not prevent other fevers being reported by Dickinson in the parish. The final infectious disease reported was that of influenza, and there were multiple deaths in the parish, with four deaths from influenza on just one day.

In about 1831 after cholera first arrived in Britain at Sunderland, there is evidence that it was not restricted to populous centres (Atkinson, 1989). Barnard Castle, County Durham, which was reasonably isolated from densely urbanised areas, was also hit with cholera. No evidence has been found specifically linking cholera to the populations living at Fewston or Wharram Percy, but this example shows that physically isolated rural areas could still be affected by the same diseases (although usually not as often or as intensely). The sparsely-populated settlements in the North Yorkshire countryside therefore protected the inhabitants to some extent.

Disease caused by occupation occurred in rural settlements as well as urban ones. In areas of sandstone and shale such as Swaledale and Grassington Moor, miners contracted "miner's consumption" or "miner's asthma" through inhaling insoluble siliceous dust (Jennings, 1967). In Swaledale between 1843 and 1857, 55 of 85 miners died due to chest diseases. Lead smelters were liable to lead poisoning

caused by absorbing the lead fumes and dust through respiration and ingestion (*ibid.*). Men were not the only ones exposed to lead as women from at least the 18th century were employed to redress lead ore from old waste heaps. During the post-medieval period, working in the textile industry evolved from working at home, to working 12-13 hours in a mill (*ibid.*). It was mostly women and children that were employed, although a few men worked as mechanics and overseers or occasionally prepared the textiles (*ibid.*). Employment in mills was 'under difficult, unpleasant working conditions' (Alred, 1997:30). Standing and attending to spinning machines or looms for long hours was responsible for the crippling effects and disfigurement to the arms, legs, and back that people experienced (Alred, 1997). Unfortunately, nothing more specific about these disfigurements was provided in Alred's (1997) account.

Another serious health problem arising from working in textile mills was byssinosis, or brown lung. This is caused by inhaling cotton or hemp dust, two common materials processed in Fewston parish (Alred, 1997; Nafees *et al.*, 2013; Tahir *et al.*, 2012). In an effort to combat this disease, the 1800 Factory Bill required mills to be ventilated with proper windows (Alred, 1997). Francis Thorpe, mill owner, stated in about 1803 'I have never seen the slightest ill-effect result...to the health of the work people from working twelve hours a day' and pointed out that there were two large ventilation areas in the tow-carding room to remove dust in an attempt to protect against lung disease (Jennings, 1967:227).

Some healthcare was available to inhabitants of rural communities in North Yorkshire, although some travel may have been involved, as seen in John Dickinson's diary (Harker, 1988). Dickinson had false teeth fitted by a dentist and both he and his daughter purchased spectacles, although he does not state whether these were acquired locally or not. His daughter Dorothy travelled to Manningham near Bradford to receive special treatment for lameness in her leg, and in Dickinson's final days, a physician and nurse visited him in his home on multiple occasions before removing him to Leeds Infirmary where he passed away.

2.3.7 Historical Literature Conclusions

Historical and archaeological sources of living environments show the presence of conditions that were detrimental to health in both urban and rural settlements. Both sets of locations potentially had difficulties with providing good quality housing, a nutritious diet, a clean water supply, and a good level of sanitation. Each context provided risk factors for health. However, it is likely that detrimental conditions were compounded in urban contexts due to the higher population densities and greater incidence of industry and its associated polluting properties. For example, while houses from both contexts may have been smoky and crowded inside, rural inhabitants would have been able to escape the closeness of other humans and coal smoke by stepping outside. Urban dwellers were not; conditions outside of the home were likely to be smokier and the tightly packed houses would have made it difficult to avoid large numbers of people. Related to this is the spread of infectious disease. As mentioned in Section 1.2.2, infectious diseases need a sizeable population of humans before the disease can become endemic. Infectious disease would have been spread into small communities through the movement of people but these were likely isolated incidents. In urban areas, infectious diseases were a constant presence. Conditions such as these and the others mentioned in this section were teratogenic (Section 2.2) and may have led to the formation of congenital defects that can be seen archaeologically. In the next section, the congenital defects visible on skeletal remains will be described.

2.4 Congenital Defect Literature Review

'Minor developmental disorders that do not threaten life are easily recognized in ancient human skeletal populations while more severe life threatening developmental disorders...rarely survive in archaeological material.'
(Barnes, 2012b:380)

As stated in Chapter 1 congenital defects may be caused by the poor living conditions detailed above (Section 2.3) due to the presence of teratogenic agents (Section 2.2). Differences in the frequencies of congenital defects may therefore

show differences in living environments. In this section, the congenital defects observed in this study are described below. Frequency rates given for defects were ascribed to nations or ethnic groups whenever possible. Unfortunately, no data specific to the Northeast of England could be found on the frequency of the defects detailed here. Images are provided of archaeological remains and modern individuals affected by the conditions whenever possible. Not all congenital defects mentioned in Section 2.2 were chosen for study due to their rarity in bioarchaeological literature (*e.g.* anencephaly or microcephaly) or location in the body (*e.g.* syndactyly or clubbed feet as examination of hands and feet was not included in this study). The congenital defects mentioned in Section 2.2 as being caused by specific known teratogens are referred back to Table 2.1 in the descriptions below.

2.4.1 Craniosynostosis

Craniosynostosis (sometimes referred to as craniostenosis) is a premature closure, typically *in utero*, of any suture of the cranium (see Table 2.1) (Barnes, 1994; Giuffra *et al.*, 2013; Sharma, 2013), with the exact mechanism bringing about this defect unknown (Bennett, 1967; Karabagli, 2013; Sharma, 2013). Investigation of the two terms used for this defect reveals that craniosynostosis is the premature fusion and craniostenosis is the resulting abnormal head shape (Duncan and Stojanowski, 2008). As the goal of this thesis is to focus on the presence of congenital defects more than the outcome of the defects, the term craniosynostosis is used to refer to premature fusion of cranial sutures. This is also in keeping with current medical literature where craniosynostosis appears to be the more popular term in describing the development and presence of these defects.

Craniosynostosis results in cranial deformity that may be symmetrical or asymmetrical (Figure 2.9). Due to the premature fusion (partial or complete), some areas of the cranium have restricted growth, leading to compensatory “bossing,” or projection, in other areas (Giuffra *et al.*, 2013; Sharma, 2013). Craniosynostosis has an incidence rate of about 1 in 2,500 live births (Giuffra *et al.*, 2013; Roscioli *et al.*, 2013).

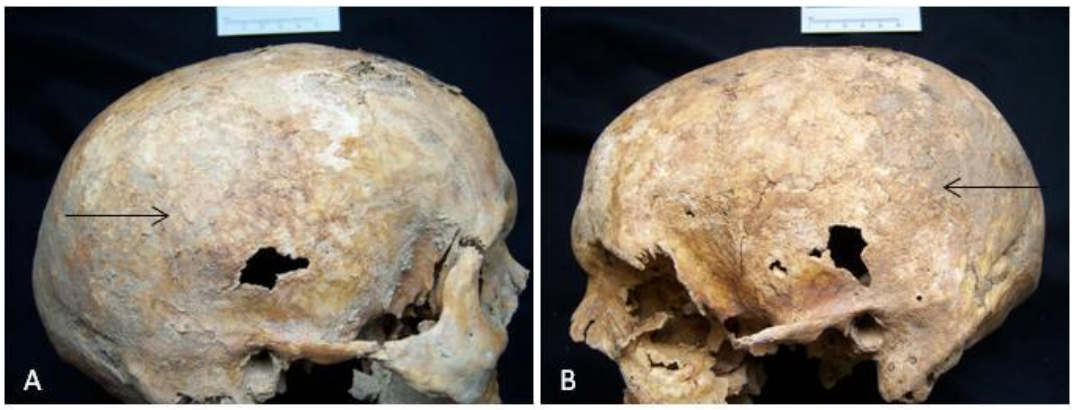

Figure 2.9: Craniosynostosis of the right squamosal and occipito-mastoid sutures in a male over 45 years old. (**A**) Showing the obliterated right squamosal suture. (**B**) Showing the present left squamosal suture. (F351, Fewston)

The most commonly affected suture is the sagittal suture, and synostosis of this suture affects more males than females (Barnes, 1994; Duncan and Stojanowski, 2008; Roscioli *et al.*, 2013). Early closure of this suture gives rise to a long and narrow cranium called scaphocephaly (Figure 2.10A) (Barnes, 1994; Kutterer and Alt, 2008; Sharma, 2013). The second most commonly affected suture is the metopic suture occurring in 1 per 5,200 births (Karabagli, 2013). Craniosynostosis of the metopic suture produces a triangular, pointed frontal bone with a narrow forehead (trigonocephaly) (Barnes, 1994; Karabagli, 2013; Sharma, 2013). Coronal synostosis occurs in 0.8-1.0 per 10,000 live births (Giuffra *et al.*, 2013). Premature fusion of the coronal suture is more common in females (60-75% of occurrences) and leads to a rounded cranium with prominent eyes and bilateral frontal bossing (brachycephaly) (Barnes, 1994; Giuffra *et al.*, 2013; Pedersen and Antón, 1998; Sharma, 2013). This early fusion leads to the orbits having an upturned appearance at their lateral edges, termed “harlequin eye,” and increased interorbital breadth (Pedersen and Antón, 1998). If the coronal suture is only partially closed (*i.e.* unilateral), the cranium is more asymmetrical (plagiocephaly) (Figure 2.10B). On the affected side, the orbital rim and the ear is high and anterior while the chin is displaced to the opposite side of the closure (Sharma, 2013). The lambdoidal suture is rarely affected but when it is present, it is affected more commonly unilaterally, and in males (Barnes, 1994; Haas-Lude *et al.*, 2013). Closure of this suture accounts for 1-3% of all single suture craniosynostoses (Haas-Lude *et*

al., 2013). Unilateral lambdoidal suture closure leads to a plagiocephalic shape with a flat occipital bone, bossing on the frontal bone, and an inferiorly and posteriorly situated ear, all on the affected side, along with facial asymmetry (Haas-Lude *et al.*, 2013; Sharma, 2013). Multiple sutures may exhibit premature closure in the same individual. Early closure of both the coronal and lambdoidal sutures increases the width and height of the cranium and is called oxycephaly (Barnes, 1994). Premature closure of the coronal and sagittal sutures results in a cranium that is pointed and tower-shaped and called oxycephaly, acrocephaly, or turricephaly (*ibid.*). Craniosynostosis, particularly more severe manifestations, may lead to increased intracranial pressure, deafness, blindness, and mental retardation (Roscioli *et al.*, 2013; Sharma, 2013). Premature fusion of the squamosal suture and unilateral fusion of the coronal suture results in plagiocephaly due to the coronal changes rather than from any involvement of the squamosal suture (Duncan and Stojanowski, 2008).

Figure 2.10: (**A**) Scaphocephaly due to craniosynostosis of the sagittal suture (Sharma, 2013). (**B**) Plagiocephaly due to unilateral craniosynostosis of the coronal suture (Sharma, 2013).

2.4.2 Cleft Lip and Palate

Cleft lip is the most common type of craniofacial clefting and the second most common severe congenital defect in humans behind talipes equinovarus, or clubfoot (Barnes, 1994; Kosowski *et al.*, 2012). A cleft lip is a separation resulting from hypoplasia or aplasia in the premaxilla (Figure 2.11, see Table 2.1) (Barnes, 1994, 2012a; Tse, 2012). These defects can be bilateral or, more commonly, unilateral. In unilateral occurrences, the cleft is most frequently seen on the left

side (*ibid.*). As the clefting occurs in the region of the maxillary incisors, the development of the teeth within the defect area will be affected. This commonly leads to agenesis of the incisors but can also lead to hypoplasia, rotation, non-eruption, malformation, or misplacement of teeth (Barnes, 1994, 2012a; Klein *et al.*, 2013). There is a range of severity expressed for this defect. The cleft may be a complete gap between the nasal and oral cavities or it may be a smaller notch that is covered by soft tissue (Barnes, 1994; Tse, 2012). Cleft lips are more common in males than in females (*ibid.*) and the incidence of cleft lip varies greatly by ancestry, occurring in 1 per 450 live births in Native American and Asian populations, 1 per 1,000 live births in Caucasian populations, and 1 per 2,000 live births in African American populations (Kosowski *et al.*, 2012; Tse, 2012). A majority of individuals with a cleft lip will also have a cleft palate, particularly in the more severe occurrences of cleft lip (Barnes, 1994, 2012a; de Aquino *et al.*, 2013; Kosowski *et al.*, 2012; Ortner, 2003). Cleft palates in these individuals arise due to developmental disruptions in the maxillary plate and the maxillary palatal plates adjacent to the abnormal premaxilla (Barnes, 1994, 2012a; Kosowski *et al.*, 2012). If the cleft lip is severe, death may occur due to suckling and breathing difficulties (Barnes, 1994; de Vries *et al.*, 2013; Ortner, 2003), while individuals with unilateral clefting have been shown to have a better chance of survival (Barnes, 2012a). Other difficulties arising from the presence of a cleft lip include upper respiratory and middle ear infections, hearing loss, and speech problems (Barnes, 1994).

Figure 2.11: Bilateral cleft lip without a cleft palate (Leslie and Marazita, 2013).

Cleft palate, separate from cleft lip, is the third most common congenital defect in humans (Kosowski *et al.*, 2012). When cleft palate occurs on its own, it is caused by hypoplasia or aplasia of one or both sides of the palate (Figure 2.12, see Table 2.1) (Barnes, 1994, 2012a; Kosowski *et al.*, 2012). The shortened growth of the palate is due to a timing delay in the descent and development of the primitive tongue, blocking the midline and hindering the growth and fusion of one or both sides (Barnes, 2012a; Leslie and Marazita, 2013). Thus, a cleft palate is an opening in the palate, allowing communication between the oral and nasal cavities (Aufderheide and Rodriguéz-Martín, 1998). As the premaxilla and maxilla are not affected by this developmental delay, the teeth are unaffected (Barnes, 1994). Cleft palates may be bilateral, appearing either symmetrical or asymmetrical, or more commonly unilateral (Barnes, 1994, 2012a; Kosowski *et al.*, 2012). The most severe clefting results from bilateral aplasia (Barnes, 1994). Cleft palate occurs in about 1 per 1,000-2,000 live births, with little variation in incidence between ancestries, and is more common in females (Aufderheide and Rodriguéz-Martín, 1998; Barnes, 1994; Kosowski *et al.*, 2012; Tse, 2012). Cleft palates may cause feeding and lower respiratory difficulties that can lead to death in the first year of life (Anderson, 1994; Aufderheide and Rodriguéz-Martín, 1998; Barnes, 1994; de Vries *et al.*, 2013; Kosowski *et al.*, 2012; Leslie and Marazita, 2013; Ortner, 2003). This defect also leads to increased upper respiratory tract infections and middle ear infections as well as hearing loss and speech problems (Barnes, 1994; Kosowski *et al.*, 2012).

Figure 2.12: Unilateral cleft palate without the presence of a cleft lip (Leslie and Marazita, 2013).

2.4.3 Elongated Styloid Process

The styloid process is a thin bony projection inferior to the external auditory meatus of the temporal bone. While the length of this process varies by population, processes longer than 3 cm are considered to be "elongated" (Figure 2.13) (Piagkou *et al*., 2009). The styloid process itself may be elongated or the increased length may be due to ossification of the stylohyoid ligament (Barnes, 2012a; Resnick and Niwayama, 1995a). When the bone element is elongated, it has a smooth contour and projects antero-inferiorly, whereas if the length is caused by ossified soft tissue, the surface will be irregular with one or more areas of thickness, and it is generally bent medially (Rajić Šikanjić and Vlak, 2010: after Skrzat *et al*., 2007). The condition usually occurs bilaterally and is more common in females (Piagkou *et al*., 2009). Past clinical studies from developed nations have shown frequency rates that range from 0.4-84.4%, although most report a rate of less than 30% (*ibid.*). The trait is largely asymptomatic but a small percentage of individuals experience Eagle's syndrome (Piagkou *et al*., 2009), which has a wide spectrum of symptoms that include ear, neck, throat, temporomandibular joint, or tongue pain, dysphagia (difficulty in swallowing), odynophagia (pain caused by swallowing), dysphonia (vocal changes), changes to taste, facial paraesthesia, and foreign body sensation in the throat (Piagkou *et al.*, 2009; Resnick and Niwayama, 1995a). These symptoms are a result of the close association of the process with the carotid artery, jugular vein, and the accessory, hypoglossal, vagus, and glossopharyngeal nerves (Resnick and Niwayama, 1995a). Archaeologically, reported elongated styloid processes are rare due to the high rate of post-mortem breakage of this thin element, but that does not mean the presence of an abnormal styloid should be ignored (Kase, 2010). While the term Eagle's syndrome has been used previously in bioarchaeological reports, it will not be used in this study as the diagnosis of the syndrome requires the presence of certain clinical symptoms which cannot be observed in an archaeological (*i.e.* dead) skeleton. Instead, the trait is called an elongated styloid process.

Figure 2.13: Right temporal bone demonstrating an elongated styloid process in a male 30-39 years old. (CS06 332, South Shields)

The aetiology of an elongated styloid process is uncertain. An anomaly of development is a likely cause but other possibilities include genetic inheritance, and endocrine dysfunction (Piagkou *et al*., 2009). Ossification of the stylohyoid ligament is often caused by trauma or tonsillectomy and is commonly seen in diffuse idiopathic skeletal hyperostosis (Piagkou *et al*., 2009; Resnick and Niwayama, 1995a).

2.4.4 Cleft Neural Arch

A developmental delay that results in hypoplastic or aplastic pedicles, laminae, or spinous processes, on one or both sides of the vertebral arch, can result in a cleft neural arch (Figures 2.14) (Barnes, 1994, 2012a; Mulhern and Wilczak, 2012; Torriani and Lourenço, 2002). Additionally, clefts can occur due to a herniation of the meninges during development. The size of the cleft will be determined by the severity of the delay. When the neural tube is involved, the clefting is referred to as either spina bifida occulta or spina bifida cystica, depending on the severity (see Table 2.1). Spina bifida cystica is the more severe form and is often fatal (Aufderheide and Rodríguez-Martín, 1998). This form leads to thin pedicles, and absent or deformed laminae and spinous processes (Barnes, 1994). The meninges, nerves, and/or spinal cord protrude through the ensuing gap and may or may not be covered by skin and other soft tissue in life (Aufderheide and Rodríguez-Martín, 1998; Ortner, 2003). In spina bifida occulta, the neural tissues do not actually extend beyond the bony structure of the arch. In both types of spina

bifida, the neural arches are pushed outward by the cystic growth and the spinal canal is wide due to the involvement of the soft tissue defect (Barnes, 1994, 2012a). Cleft neural arches without neural tube involvement are not raised and the spinal canal is of normal width because these are only caused by bone defects (Figure 2.15).

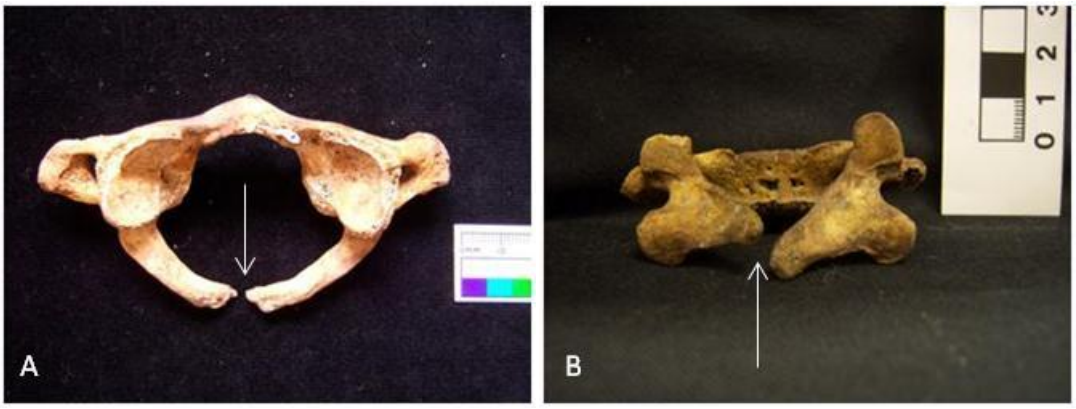

Figure 2.14: (**A**) Cleft neural arch of the first cervical vertebra in a male over 40 years old. (F366, Fewston) (**B**) Cleft neural arch of the fifth lumbar vertebra 4-7 year old non-adult. (COL10 199S, North Shields)

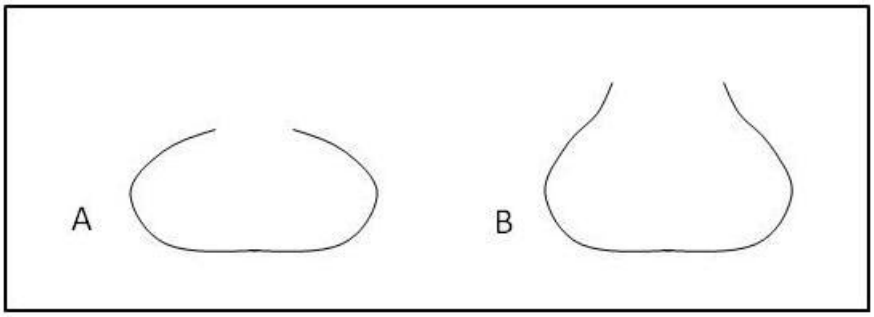

Figure 2.15: Line drawing of the anatomy of the vertebral canal and neural arch in a cleft neural arch (**A**) and spina bifida occulta (**B**).

There is much confusion over the terms used for the varying clefts that are observed in both clinical and palaeopathological settings where often “spina bifida occulta” or “spina bifida” are used as general terms for all clefts (Barnes, 1994, 2012a; personal observation). This, however, is technically incorrect as spina bifida refers to a developmental defect involving the neural tube, but not all clefts involve these soft tissues. Ozonoff states,

> 'The use of the term "spina bifida occulta" for these minor failures of fusion [clefts without neural tube involvement] is to be strongly discouraged as the term implies significant clinical abnormalities, which generally are not present. The term "spina bifida", if it is to be used at all, should be restricted to those instances of severe dysraphism [incomplete fusion] with neurologic consequences' (1995:4246).

Meanwhile, Barnes advises that:

> 'If the difference can be determined, reference should be made to spina bifida occulta resulting from neural tube defect versus cleft neural arch defect instead of calling both spina bifida occulta. This would help to determine the true incidence of neural tube defects' (1994:49).

Cleft arches occur most commonly in the lumbosacral region but about 5% of modern adults exhibit clefting at the first cervical vertebra (Aufderheide and Rodríguez-Martín, 1998; Barnes, 1994; Conner and Ferguson-Smith, 1997; Dickel and Doran, 1989). Clefting without involvement of the neural tube usually affects only one or two vertebral segments and are typically asymptomatic. Occurrences without neural tube involvement are much more common than those with, and can appear in up to 25% of the population (Barnes, 1994). In spina bifida, depending on the severity of the defect, symptoms can include lower back and leg pain, paralysis, foot deformities, hydrocephalus, and kyphoscoliosis (Aufderheide and Rodríguez-Martín, 1998; Barnes, 1994; Conner and Ferguson-Smith, 1997; Dickel and Doran, 1989; Resnick, 1995a). In severe instances, it can prove to be fatal.

2.4.5 Hypoplastic and Aplastic Lamina

Disturbances to the cartilaginous preforms of the neural arch or developmental delays in the ossification of the neural arch, can lead to aplasia or hypoplasia of the laminae of the arch or sacral crest (Barnes, 2012a; Mellado *et al.*, 2011a; Torriani and Lourenço, 2002). Aplasia is the complete absence of one or both sides of the lamina, while hypoplasia is the reduced size of the bony arch. Aplastic neural arches cause clefting of the neural arch as the two sides of the

lamina are not joined at the midline because one or both are absent. The space is bridged by fibrous tissue in life (Torriani and Lourenço, 2002; Villas and Barrios, 1997). Bilateral aplasia may be asymptomatic but can lead to kyphoscoliosis (antero-posterior and lateral curvatures of the spine), neurological impairment, and pain (Torriani and Lourenço, 2002; Villas and Barrios, 1997). Hypoplastic neural arches can result in cleft neural arches but can also lead to intact arches depending on the rate of developmental delay (Figure 2.16). As cleft neural arches caused by hypoplasia of the laminae have already been discussed (Section 2.4.4), this section focuses on hypoplastic neural arches without clefting. In bilateral occurrences, the neural arch has a gracile appearance and the condition is mainly asymptomatic but may cause stenosis, or narrowing, of the spinal canal (Barnes, 1994, 2012a; Torriani and Lourenço, 2002; Van Roy *et al.*, 1997). Hypoplastic neural arches without clefting are most commonly seen in the atlas. In unilateral occurrences, the neural arch has an asymmetrical appearance. This can lead to uneven load bearing in the lumbar spine, stenosis of the spinal canal, and slight curvatures or scoliosis at any affected location in the spine (Barnes, 2012a; Brash, 1915; Torriani and Lourenço, 2002).

Figure 2.16: Hypoplastic lamina unilateral (left side) of the third cervical vertebra in a non-adult about 12 years old. (CS06 678, South Shields)

2.4.6 Spondylolysis

A lack of union of the vertebra at the pars interarticularis (the area just inferior to the superior articular facets) is termed spondylolysis (Figure 2.17) (Aufderheide and Rodríguez-Martín, 1998; Merbs, 2002; Ortner, 2003; Pilloud and

Canzonieri, 2012; Resnick *et al*., 1995). This creates two separate portions of the bone: the anterior (containing the body, pedicles, transverse processes, and superior articular facets) and the posterior (containing the laminae, spinous process, and inferior articular facets). The defect can be either bilateral where the neural arch is completely separate or, less commonly, unilateral so that the arch is still attached on one side (Barnes, 2012b; Merbs, 2002). The lumbar region is most frequently involved, often at the fifth vertebra (in 67% of occurrences, followed by L4 at 15-30%, and then the L3 at 1-2%) (Pilloud and Canzonieri, 2012; Resnick *et al*., 1995). Spondylolysis can also occur in multiple vertebral segments within one individual. Around 2-10% of the population is affected, males slightly more so than females, although among certain families and ethnic groups (*e.g.* Inuits) the prevalence of spondylolysis can be as high as 69% (Aufderheide and Rodríguez-Martín, 1998; Merbs, 1996; Pathria, 1995; Resnick *et al*., 1995). While often asymptomatic, spondylolysis may cause back pain, gait abnormalities, and neurological problems (Pathria, 1995; Resnick *et al*., 1995). Additionally, it may increase flexibility in the lower back (Merbs, 1989, 1996). It should be noted that spina bifida occulta can accompany spondylolysis in many individuals (Pathria, 1995; Resnick *et al*., 1995; Yamada *et al.*, 2013).

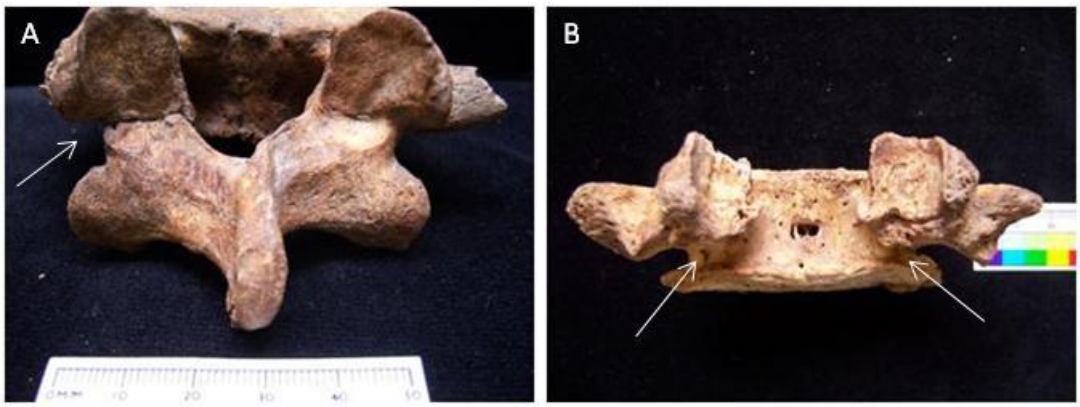

Figure 2.17: (**A**) Left unilateral spondylolysis of the fifth lumbar vertebra in a male 25-34 years old. (COL10 115, North Shields) (**B**) Bilateral spondylolysis of the fifth lumbar vertebra in a male over 40 years old. The neural arch was lost post-mortem. (F366, Fewston)

The aetiology of spondylolysis is uncertain and has been debated since at least 1884 (Fibiger and Knüsel, 2005; Pilloud and Canzonieri, 2012; Yamada *et al.*, 2013). It has been attributed to a stress fracture, congenital weakness, or a

combination of the two. An additional associated feature is curvature of the lower spine associated with bipedal locomotion and erect posture (Farfan *et al*., 1976; Fibiger and Knüsel, 2005; Merbs, 1989, 1996). It has been suggested that a congenital cause can be assumed if other spinal anomalies, especially aplastic or hypoplastic pedicles, are present (Merbs, 1996). Additionally, facets showing signs of a synovial joint suggest a congenital origin (Mays, 2007a). The articulating surfaces of synovial joints are encased in capsules containing synovial fluid. Synovial joints are not generated around traumatically created joints; they are only formed during development, so a "synovial presence" indicates that a joint was there from embryonic development. The location of the separation has been argued to play a part in the determination of its aetiology where acquired spondylolysis occurs below the third lumbar vertebra and a developmental cause is responsible above L3 (*ibid.*). At the third lumbar vertebral level, spondylolysis can be due to either cause. Alternatively, all occurrences in the lumbar region may be trauma-related while a location elsewhere in the spine would indicate a congenital cause.

One argument against a congenital cause is the absence of spondylolysis reported for infants (Aufderheide and Rodríguez-Martín, 1998; Mays, 2007a), although Resnick *et al*. (1995) has reported infants as young as three months with the condition. Furthermore, in some individuals, it appears to have a hereditary cause (Pathria, 1995; Pilloud and Canzonieri, 2012; Yamada *et al.*, 2013). Out of this confusing jumble of theories, the literature seems to be reaching a consensus that there is an underlying congenital weakness of the vertebra that leads to the fracturing or separating of the two portions of the vertebra. As Merbs explains, 'it is clear that what is being inherited is not the lysis itself, but traits which predispose to its occurrence (1989:166).' Farfan *et al*. (1976) also postulates the underlying developmental cause may be elongated transverse processes. The length of the processes limits mobility around the affected vertebrae, leading to intense forces being placed on the soft tissues attached to the transverse processes. This leads to the separation of the two portions of the vertebra. Ward *et al*. (2010) propose the space between the facets, sacral inclination, and soft tissue variations such as muscle tone and sufficient vascularisation may be factors in developing

spondylolysis. Whatever the exact anatomy of the vertebra that leads to the development of this separation, spondylolysis is now believed to have a congenital cause and as such is included in this study.

2.4.7 Block Vertebrae/Klippel-Feil Syndrome

Block vertebrae, the fusion of two or more vertebrae at any location in the pre-sacral spine, are the result of the improper arrangement of vertebral segment ossification centres during development (Figure 2.18, see Table 2.1) (Olufemi Adeleye and Olusola Akinyemi, 2010; Ortner, 2003; Pany and Teschler-Nicola, 2007; Samartzis *et al.*, 2008; Usher and Christensen, 2000). Klippel-Feil syndrome was originally described as the fusion of two or more cervical vertebrae combined with a webbed neck and low hairline (Barnes, 1994, 2012a; Ozonoff, 1995; Pany and Teschler-Nicola, 2007; Resnick, 1995b; Samartzis *et al.*, 2008; Silva and Ferreira, 2008). Klippel-Feil syndrome occurs as three types: Type I affecting several cervical and upper thoracic vertebrae (often associated with other anomalies), Type II affecting only two or three cervical vertebrae, and Type III affecting the cervical spine and having associated segmental errors in the thoracic and/or lumbar region (often associated with other anomalies and commonly fatal) (Barnes, 1994; Pany and Teschler-Nicola, 2007). Over time, the term “Klippel-Feil syndrome” has come to mean the fusion of cervical vertebrae (and sometimes at other locations in the spine) even without the presence of the soft tissue anomalies in both the clinical and palaeopathological literature (Olufemi Adeleye and Olusola Akinyemi, 2010; Silva and Ferreira, 2008). For the sake of this thesis, the term Klippel-Feil syndrome will be used for fusion occurring in the cervical region, and block vertebrae when occurring in the lower spine, to follow the precedence set out in the clinical literature.

Figure 2.18: Klippel-Feil syndrome resulting in fusion of the C6 and C7. (**A**) Posterior view. (**B**) Anterior view. (**C**) Left lateral view (COL 10 128, North Shields).

Block vertebrae are rare but can occur in up to 0.5% of clinical spinal radiographs (Resnick, 1995b). Fusion of this type is found equally in both sexes (Resnick, 1995b) or slightly more commonly in females (Aufderheide and Rodríguez-Martín, 1998). When present, the most commonly occurring block vertebrae are those termed Klippel-Feil syndrome Type II, most frequently affecting the second and third, or fifth and sixth, cervical vertebrae (Barnes ,1994; Resnick, 1995b). Type II occurrences and those occurring in the thoracic and lumbar vertebrae are generally asymptomatic. Block vertebrae are often associated with other anomalies, including scoliosis or kyphosis, cleft neural arch, cleft palate, cervical ribs, hemivertebrae, cranial asymmetry, brain defects, supernumerary lobes of the lung, and renal anomalies (Aufderheide and Rodríguez-Martín, 1998; Resnick, 1995b; Samartzis *et al.*, 2008). Additionally, there may be limited mobility of the neck or back and neural impingement (Ozonoff, 1995).

2.4.8 Transitional Vertebra

A transitional vertebra is one that takes on the appearance of a vertebra from an adjacent location in the spine, and arises due to segmentation errors of the sclerotomes during development of the vertebral column (Barnes, 1994, 2012a;

Khamanarong *et al.*, 2013; Mudaliar *et al.*, 2013; Senator and Gronkiewicz, 2012; Usher and Christensen, 2000). These changes are also referred to as border shifts. The shift can move in either a cranial or caudal direction. A cranial shift occurs when the vertebra takes on the appearance of the spinal elements below it, while a caudal shift occurs when the vertebra has the appearance of the elements above it (Barnes, 1994, 2012a). These shifts are named after the location and direction of the shift: occipital vertebra (occipitocervical border, cranial shift), occipitalization (occipitocervical border, caudal shift), lumbarization (thoracolumbar border, cranial or lumbosacral border, caudal), and sacralization (lumbosacral border, cranial). Additional ribs may develop due to shifts at the borders of the thoracic vertebrae. More than one shift can occur within the spine of an individual. These multiple shifts can be in the same direction or a mixture of cranial and caudal shifts since each border has its own genetic "command" to follow unrelated to what has occurred at other borders (Barnes, 2012b).

A. Occipitocervical Border

The occipitocervical border includes the occipital bone of the cranium and the first two cervical vertebrae (Barnes, 2012a). Cranial shifting is an attempt by the cranial base of the occipital bone to separate from the rest of the cranium (Anderson, 1996; Barnes, 2012a). This results in what is termed an occipital vertebra. The "vertebra" never actually separates from the occipital bone, leading to a range of appearance ranging from a protruding tubercle arising from the foramen magnum (precondylar process) to a raised ring around the foramen magnum resembling a first cervical vertebra (Anderson, 1996; Barnes, 2012a, b). Caudal shifting at this location is termed occipitalization and is an assimilation of the first cervical vertebra into the base of the cranium (Figure 2.19A, see Table 2.1) (Barnes, 1994, 2012a; Bašić *et al.*, 2012; Keenleyside, 2012a; Khamanarong *et al.*, 2013; Mudaliar *et al.*, 2013; Senator and Gronkiewicz, 2012). This may occur unilaterally or bilaterally, partially or completely. Complete occipitalization is the most common expression (Khamanarong *et al.*, 2013; Mudaliar *et al.*, 2013). Occipitalization is the most common defect at this site of the spine but is only seen

in 0.08-3.63% of modern populations from developed nations (Bašić *et al.*, 2012; Keenleyside, 2012a; Khamanarong *et al.*, 2013; Senator and Gronkiewicz, 2012). A mild expression of caudal shifting at the occipitocervical border is the presence of a precondylar facet (Figure 2.19B) (Barnes, 1994, 2012a, b; Masnicová and Beňuš, 2003). This forms when a shift delays the descending of the dens of the second cervical vertebra, causing it to protrude into the foramen magnum, creating a facet on the anterior border of the foramen. There may also be an additional facet on the first cervical vertebra. Occipitalization is commonly asymptomatic but may also result in headaches, neck pain, paraesthesia, pain in the limbs, and restricted head movement (Bašić *et al.*, 2012; Keenleyside, 2012a; Mudaliar *et al.*, 2013).

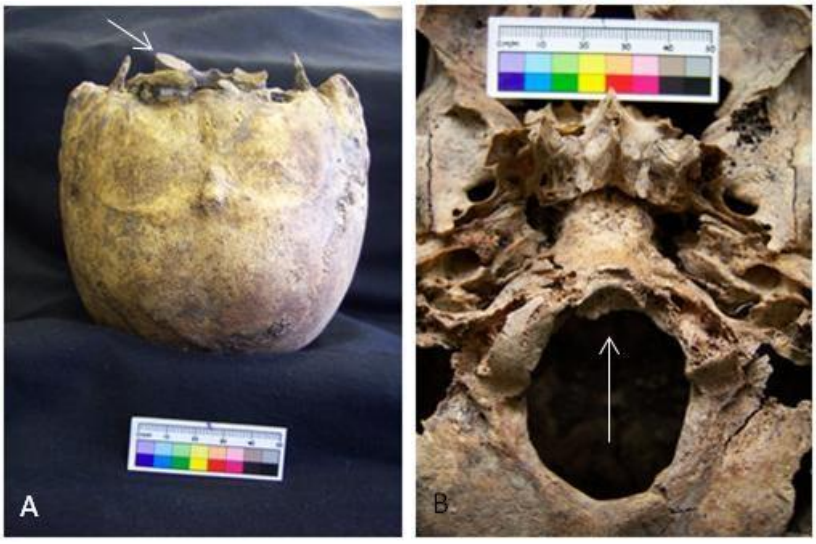

Figure 2.19: (**A**) Occipitalization of the first cervical vertebra in a male over 45 years old. (COL10 125, North Shields) (**B**) A precondylar facet, a mild expression of caudal shifting at the occipitocervical border, on the occipital bone in a female over 45 years old. (F122, Fewston)

B. Cervicothoracic Border

An elongation of the transverse process of a cervical vertebra, normally the seventh, resulting from a shift at the cervicothoracic border is known as a cervical rib (Aufderheide and Rodríguez-Martín, 1998; Barnes, 1994, 2012a) (Figure 2.20). These defects occur in 0.2-8% of modern individuals, based on clinical chest radiographs from developed nations (Walden *et al.*, 2013). The "rib" varies greatly in size and ranges from a tubercle at the end of the process to a true rib, demonstrating the anatomy of a thoracic rib, including a head, neck, body, and

often a costal joint. A cervical rib seldom reaches the sternum but is likely to articulate with the first thoracic rib. These supernumerary ribs are usually symmetrical, bilateral, and more common in females (Aufderheide and Rodríguez-Martín, 1998; Barnes, 1994; Black and Scheuer, 1997; Resnick, 1995b).

Figure 2.20: Rudimentary right cervical rib of the seventh cervical vertebra in a non-adult 10-11 years old. The left side was broken post-mortem. (F062, Fewston)

While having a cervical rib can be asymptomatic, in about a third of occurrences the rib can exert pressure on the subclavial artery and vein or brachial plexus (Barnes, 1994, 2012a; Black and Scheuer, 1997; Walden *et al.*, 2013). This can lead to pain in the neck or shoulder, paraesthesia in the hand or fingers, muscle weakness, muscle atrophy, paralysis, and a reduced pulse.

C. Thoracolumbar Border

Shifting in the cranial direction at the thoracolumbar border results in lumbarization of the twelfth thoracic vertebra (Figure 2.21) (Barnes, 1994, 2012a; Usher and Christensen, 2000). This manifests in rudimentary or absent ribs and accompanying costal facets (see Table 2.1). The apophyseal facets are also rotated to match those seen in a lumbar vertebra, *i.e.* the superior apophyseal facets face medially rather than posteriorly and have a more curved appearance.

Figure 2.21: Lumbarization of T12 (right) with accompanying changes in T11 (left) in a male 17-25 years old. The superior apophyseal facets of T12 (white arrow) are rotated to face medially instead of posteriorly while the inferior apophyseal facets of T11 (red arrow) face laterally instead of anteriorly. (COL10 015, North Shields)

Less common than cervical ribs in clinical studies, lumbar ribs arise from a caudal shift at the thoracolumbar border where the transverse processes are elongated (Figure 2.22, see Table 2.1). They are variable in size from a tubercle-like projection to a true articulating rib (Barnes, 1994, 2012a; Resnick, 1995b). Lumbar ribs are typically bilateral, although commonly asymmetrical, found on the first lumbar vertebra, have associated costal facets, and are more common in females (Barnes, 1994). The presence of these ribs is generally asymptomatic, though there may be pain in the affected area (Barnes, 1994, 2012a; Resnick, 1995b).

Figure 2.22: Bilateral lumbar ribs of the L1 in a female over 45 years old. Costal facets were present bilaterally but only the right lumbar rib was excavated. Additionally, the left superior articular facet faces posteriorly (arrow). (F363, Fewston)

D. Lumbosacral Border

Transitional vertebrae at the lumbosacral border are common, occurring in 4-36% of modern populations from developed and developing nations and may be associated with lower back pain and curvatures of the spine (Apazidis *et al.*, 2011; Barnes, 2012a; Hughes and Saifuddin, 2004, 2006; Mahato, 2010a). Lumbarization is the partial or complete separation of the first sacral segment of the sacrum and is the term given to caudal shifting at the lumbosacral border (Figure 2.23A) (Apazidis *et al.*, 2011; Barnes, 1994, 2012a; Mahato, 2010a; Masnicová and Beňuš, 2003). The alae of this sacral segment take on the appearance of lumbar transverse processes. Lumbarization is seen in about 5.5% of individuals in modern populations (Mahato, 2010a). In the direction opposite to lumbarization, cranial shifting at the lumbosacral border is termed sacralization (Figure 2.23B). This occurs when the lowest lumbar vertebra completely or incompletely joins the sacrum, and the transverse processes are ala-like in appearance (Apazidis *et al.*, 2011; Barnes, 1994, 2012a; Hughes and Saifuddin, 2006; Mahato, 2010a, b; Masnicová and Beňuš, 2003). Sacralization occurs in 7.5% of individuals in modern populations (Mahato, 2010a). Sacralization may be partial, and here the transverse processes articulate with the alae, incomplete where only part or one side of the lumbar vertebra assimilates with the sacrum, or complete where the whole lumbar vertebra assimilates entirely with the sacrum (Barnes, 1994; Mahato 2010a, b).

Figure 2.23: (**A**) Complete lumbarization of the first sacral segment in a male 35-39 years old. (COL10 176, North Shields) (**B**) Partial sacralization of the fifth lumbar vertebra shown with the sacrum in a female 40-44 years. (CN41, Wharram Percy)

2.4.9 Numerical Variation in Vertebrae

While the normal pre-sacral spine is made up of 24 vertebrae, divided into three types (seven cervical, twelve thoracic, and five lumbar), variation can occur in the number of elements present. As discussed in Section 2.4.8, in occurrences of transitional vertebrae, the number of each type of vertebra can vary, while keeping the total number for all pre-sacral elements at 24. In other individuals, variation in the number of somites (precursors to vertebrae) occur (Barnes, 1994, 2012a; Bornstein and Peterson, 1966; de Beer Kaufman, 1974; Ibrahim *et al.*, 2013; Usher and Christensen, 2000). This leads to a true addition or subtraction to the overall number of elements present, resulting in 23 or 25 pre-sacral vertebrae present, or 28 or 30 vertebral elements when including the sacrum (see Table 2.1). A reduction in the number of vertebral elements is rare while an increase is much more common (Barnes, 1994, 2012a). Additional vertebral segments are most commonly found in the lumbar region and when present, the sixth lumbar vertebra is often sacralized (see Section 2.4.8.D). Occasionally, an additional sacral segment is seen but additional cervical or thoracic elements are rare (Barnes, 1994, 2012a; Brash, 1915).

2.4.10 Scoliosis

Scoliosis is defined as an abnormal lateral curvature of the spine (Figure 2.24, see Table 2.1) (Giampietro *et al.*, 2003; Ortner, 2003). This defect is not limited to a lateral curvature but is also present with rotation of the vertebrae towards the convexity of the curve (Couoh, 2013; Ortner, 2003; Wever *et al.*, 1999). The curvature is often present as a double or "s" curve, allowing the head to stay close to the mid-sagittal line (Ortner, 2003). Severe curvatures can result in compromised respiratory function, particularly in individuals with multiple congenital defects of the spine (Giampietro *et al.*, 2003; Redding *et al.*, 2008). Scoliosis can also affect many areas of the body aside from the vertebrae. Curves in the thoracic vertebrae lead to deformities of the ribs ("rib hump"), where the rib angle is either increased or decreased depending on the side of the ribs affected and the direction of the curve (Kilgore and Van Gerven, 2010; Ortner, 2003; Wever *et al.*, 1999). The scapula on the convex side of the curve is commonly elevated (Kilgore and Van Gerven, 2010), and occasionally, distortions of the pelvis are seen due to asymmetrical muscle action and growth. This is seen as an elevation of the pelvis on the concave side of the curve and can affect locomotion (Couoh, 2013; Kilgore and Van Gerven, 2010).

Figure 2.24: Left-sided curvature of scoliosis in T3-T7 in an adult male. (CS06 315, South Shields)

Two types of scoliosis, congenital and idiopathic scoliosis, were considered to fall under the remit of this study as other types had aetiologies unrelated to congenital causes. Congenital scoliosis accounts for around 15% of all scoliosis in patients and arises from the presence of a congenital defect occurring in one or more vertebrae (Giampietro *et al.*, 2003; Kilgore and Van Gerven, 2010; Ozonoff, 1995). The defect can be a segmentation error (*e.g.* hemivertebra) or a formation defect (*e.g.* agenesis of one side of a neural arch) that forces the rest of the spine into a curved position to compensate for the uneven vertebra, combined with different growth potentials occurring on each side of the spine (Aufderheide and Rodriguéz-Martín, 1998; Barnes, 1994, 2012a; Giampietro *et al.*, 2003; Kilgore and Van Gerven, 2010; Ozonoff, 1995). Congenital scoliosis is rare, appearing in 0.5-1.0 of 1000 live births in modern populations from developed countries (Giampietro *et al.*, 2003).

Idiopathic scoliosis cannot be attributed a cause but its aetiology is thought to arise from a neuromuscular asymmetry in the muscles of the trunk leading to differential growth on each side of the spine (Burwell, 1998; Giampietro *et al.*, 2003). Idiopathic scoliosis accounts for around 90% of scoliosis occurrences and has a frequency of 0.2-3.0% in modern populations (Figure 2.25) (Burwell, 1998; Giampietro *et al.*, 2003; Sud and Tsirikos, 2013). This form of scoliosis is up to ten times more common in females than in males and females are more likely to have progressive and clinically significant spinal curves (Burwell, 1998; Ozonoff, 1995). Sub-classification of idiopathic scoliosis is based on the age the curvature starts to form or become noticeable (Giampietro *et al.*, 2003; Ozonoff, 1995; Sud and Tsirikos, 2013): infantile scoliosis is detected in individuals under the age of three, juvenile scoliosis in individuals between three and eleven years, and adolescent scoliosis in those over the age of eleven. Of these, adolescent scoliosis is the most common sub-classification (Burwell, 1998).

Figure 2.25: Non-adult with a severe right thoracic idiopathic scoliosis (Sud and Tsirikos, 2013).

2.4.11 Rudimentary and Aplastic Transverse Process

The transverse processes of any vertebral segments may be rudimentary or hypoplastic due to a delay in development (Figure 2.26) and can occur unilaterally or bilaterally (Barnes, 1994, 2012a). Additionally, the process may not form at all, leading to an absent or aplastic transverse process (*ibid.*).

Figure 2.26: Bilateral rudimentary transverse processes on T1 (right vertebra, arrows point to affected processes) compared to normal T2 (left vertebra) from the same individual, a male 40-44 years old. (V42, Wharram Percy)

2.4.12 Rudimentary and Aplastic Apophyseal Facet

Rudimentary or hypoplastic and aplastic apophyseal or articular facets are likely to be caused by a delay in ossification and can be seen unilaterally or bilaterally (Figure 2.27) (Barnes, 1994, 2012a; Mellado, *et al.* 2011b; Shim and Oh, 2008). The presence of a rudimentary or aplastic apophyseal facet on one vertebra

typically affects the development of the complimentary facet on the adjoining vertebra (Barnes, 2012a). In unilateral appearances, commonly seen at the lumbosacral border, the joint is usually affected by uneven loading and a conjoined nerve root leads to back pain (Barnes, 1994; Mahato, 2010c; Mellado *et al.*, 2011b; Shim and Oh, 2008). A rudimentary or aplastic apophyseal facet often occurs with hypoplasia of the lamina but can also occur on its own (Barnes, 2012a; Shim and Oh, 2008). When this defect occurs at the lumbosacral border, it may be associated with the presence of an accessory facet or partial sacralization (Mahato, 2010c).

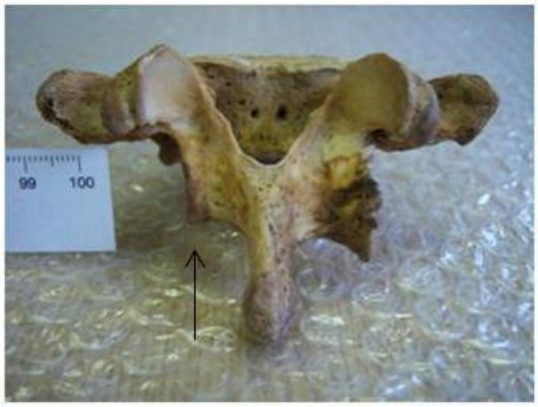

Figure 2.27: Left inferior rudimentary apophyseal facet of the fifth lumbar vertebra in a female 35-39 years old. (CS06 956, South Shields)

2.4.13 Facet Tropism

Facet tropism is asymmetry between apophyseal facet angles on the same vertebra, particularly in the lumbar region (Figure 2.28) (Chadha *et al.*, 2013; Kim *et al.*, 2013; Lee *et al.*, 2006; Linov *et al.*, 2013; Mellado *et al.*, 2011b; Van Roy *et al.*, 1997). In other words, the apophyseal facets on one side of a vertebra face in a different direction to the other (*e.g.* the right superior apophyseal facet faces medially while the left superior apophyseal facet faces posteriorly) due to a rotation of one of the facets. The articulating facet on the adjacent vertebra will also be affected. Reports of the clinical symptoms of this defect are opposing. It may be asymptomatic or it may lead to disc herniation or osteoarthritis at the affected joint (Chadha *et al.*, 2013; Grogan *et al.*, 1997; Kim *et al.*, 2013; Lee *et al.*, 2006; Linov *et al.*, 2013; Mellado *et al.*, 2011b).

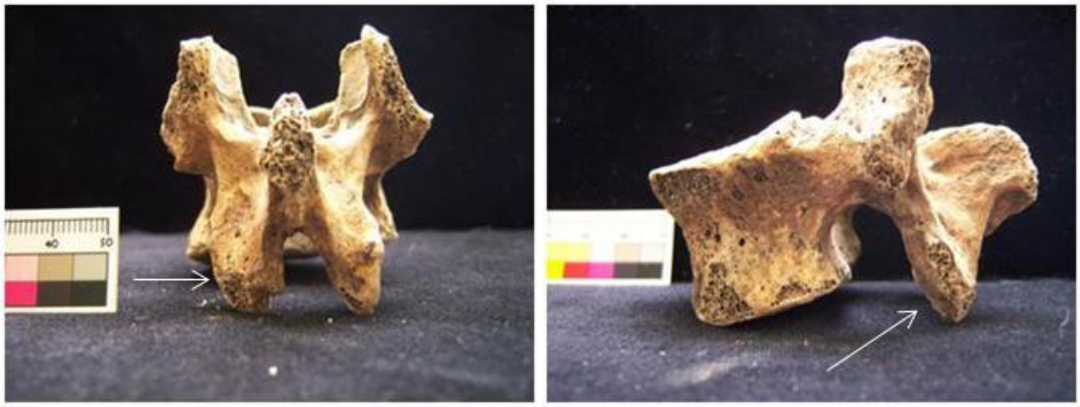

Figure 2.28: Facet tropism of the first lumbar vertebra of a male 25-34 years old. The left inferior apophyseal facet is rotated abnormally to face anteriorly (as shown by the arrows in both pictures). (COL10 210, North Shields)

2.4.14 Supernumerary Ribs

The term supernumerary rib refers to the occurrence of thirteen or more ribs on one or both sides of the thoracic cavity (Barnes, 2012a). This definition would therefore include cervical and lumbar ribs. For clarity in this thesis, cervical and lumbar ribs are treated separately (Sections 2.4.8.B and 2.4.8.C) and the term supernumerary rib applies only to other instances of thirteen ribs in an individual (*i.e.* intrathoracic ribs and supernumerary vertebrae). Intrathoracic ribs are rare, arising due to a segmentation error and are most common on the right side (Barnes, 2012a; Basarslan *et al.*, 2012; Bottosso and Ghaye, 2008; Chung and Pipavath, 2009; Kamano *et al.*, 2006). The supernumerary rib develops parallel to adjacent ribs or grows inferiorly (Barnes, 2012a; Bottosso and Ghaye, 2008). The presence of a supernumerary rib is usually asymptomatic but may cause pain or respiratory problems if it has a fibrous band attached to the diaphragm (Barnes, 2012a; Bottosso and Ghaye, 2008; Chung and Pipavath, 2009). When a supernumerary vertebra occurs in the thoracic region, a thirteenth set of ribs, formed in the same way as the other ribs in the thoracic cavity, may be associated with it (Barnes, 2012a; Brash, 1915; Usher and Christensen, 2000). These ribs arise through the same pathways as normal ribs but are commonly rudimentary and can be bilateral or unilateral.

2.4.15 Other Rib Anomalies

Congenital rib anomalies (including the cervical and lumbar ribs already discussed in Sections 2.4.8B and 2.4.8C) occur in 0.15-3.4% of the modern population with higher rates reported for a study of Samoans (10.4%) and are twice as common in males as in females (Basarslan *et al.*, 2012; Cosson *et al*., 2012). Rib defects are generally asymptomatic and are usually found in modern patients only fortuitously through radiography undertaken as a result of complaints unrelated to the rib anomaly (Allwyn Joshua *et al.*, 2013; Cosson *et al.*, 2012). Ribs develop from the mesoderm growing ventrolaterally from the chondrification sites at the costal facets of the vertebrae (Barnes, 1994; Cosson *et al.*, 2012; Wattanasirichaigoon *et al.*, 2003).

Segmentation errors occurring during the formation of the ribs take place mostly at the sternal ends and can lead to various defects including fused, broad, bridged, and bifid ribs, as well as rib spurs (Barnes, 1994; Ortner, 2003; Wattanasirichaigoon *et al.*, 2003). Fused ribs are defined as the union of two neighbouring ribs and occur most frequently between the first and second ribs (Figure 2.29A, see Table 2.1) (Aufderheide and Rodriguéz-Martín, 1998; Barnes, 1994, 2012a; Resnick, 1995b). A broad rib gives the appearance of two conjoined ribs due to its width but it is actually a single rib that is abnormally wide (Figure 2.29B) (Barnes, 2012a; Martin, 1960). Bridged ribs are similar to fused ribs but only a small osseous bridge connects the two ribs (Figure 2.29C) (Barnes, 1994). In some occurrences, the bridging is incomplete, leading to an articulation between the two ribs rather than an osseous attachment (Barnes, 1994; Kaneko *et al.*, 2012). (It should be noted that Kaneko *et al.*, 2012 diagnosed one “case study” with a bifid rib but the description and image appear to show a bridged rib.) Bifid or bifurcated ribs are forked at the sternal end with the two ends sharing one cartilage (Figure 2.29D) (Barnes, 1994, 2012a; Kaneko *et al.*, 2012; Martin, 1960; Ortner, 2003; Resnick, 1995b). This defect occurs most commonly in the third, fourth, and fifth ribs on the right side (Barnes, 1994, 2012a; Ortner, 2003) and occurs in 1.2% of the modern population (Allwyn Joshua *et al.*, 2013). In most cases, the affected rib is broader than the surrounding ribs (Ortner, 2003; Wattanasirichaigoon *et al.*, 2003). Finally, a

rib spur is a bony spike near the sternal end of a rib that may appear like an incomplete bifurcation (Figure 2.29E) (Barnes, 1994, 2012a; Martin, 1960).

Figure 2.29: (**A**) Fused ribs in a possible male 35-44 years old. (COL10 056, North Shields) (**B**) Broad rib (middle) in a non-adult roughly one year old. (COL10 131, North Shields) (**C**) Bridged ribs in a male over 45 years old. (F360, Fewston) (**D**) Bifid rib in a non-adult about three years old, with the arrow indicating the separation between the two sternal ends. (COL10 156, North Shields) (**E**) Rib spur in a male 40-44 years old. (CN45, Wharram Percy)

2.4.16 Pectus Carinatum

Pectus carinatum, commonly called pigeon chest, is the anterior bowing of the sternum (Figures 2.30 and 2.31). This arched shape is caused by abnormal growth of the ribs and costal cartilage, pushing the middle of the sternal body

forward (Barnes, 2012a; Lee *et al.*, 2013). While the defect may be visible from birth, it is most noticeable at times of rapid growth, such as early childhood or puberty, and occurs more commonly in males (8:2) (Allwyn Joshua *et al.*, 2013; Groves *et al.*, 2003; Lee *et al.*, 2013). The protrusion can by symmetrical or asymmetrical and increases the diameter of the chest. The presence of this defect is largely asymptomatic but in more severe instances, respiratory problems can arise (Allwyn Joshua *et al.*, 2013; Groves *et al.*, 2003; Lee *et al.,* 2013). The aetiology of this defect is unknown but is seen to run in families in 25% of occurrences (Allwyn Joshua *et al.*, 2013; Barnes, 2012a; Groves *et al.*, 2003).

Figure 2.30: Pectus carinatum resulting in anterior/posterior bowing of the sternum in a female over 45 years old. (CS06 107, South Shields)

Figure 2.31: Pectus carinatum in a non-adult (Lee *et al.*, 2013).

2.4.17 Os Acromiale

The acromion forms from two separate ossification centres, the epiphyseal centre and the basal part arising from the spine of the scapula. Os acromiale occurs when these two centres fail to fuse before about 25 years old, maintaining a

separate triangular-shaped acromion (Figure 2.32) (Barnes, 2012a, b; Case *et al.*, 2006; Frizziero *et al.*, 2012; Kumar *et al.*, 2013; Resnick, 1995b; Scheuer and Black, 2000). In os acromiale a synchondrosis with the acromion usually forms. This condition is often asymptomatic but can result in pain and tears to the rotator cuff (Bedi and Rodeo, 2009; Resnick, 1995b; Resnick and Niwayama, 1995b; Scheuer and Black, 2000). This defect occurs in 1.4-15% of individuals in modern populations from developed countries (Case *et al.*, 2006; Frizziero *et al.*, 2012; Kumar *et al.*, 2013). Os acromiale is bilateral in 41-62% of those occurrences (Frizziero *et al.*, 2012).

Figure 2.32: Unilateral os acromiale of the right scapula in a possible male 40-44 years old. The separate acromion is indicated by the arrow. (CS06 447, South Shields)

The aetiology of this condition is uncertain, with some researchers suggesting the separation is due to occupational stress at the joint, or an un-united fracture (Miles, 1994; Scheuer and Black, 2000; Stirland, 2000). While it would be impossible to differentiate between a traumatic and developmental cause without knowing the individual's medical history, due to the common bilateral appearance, the frequency of occurrence, and the formation of a synovial joint, this defect appears more likely to have a developmental origin and is included in this study (Case *et al.*, 2006; Frizziero *et al.*, 2012; Kumar *et al.*, 2013; Miles, 1994; Resnick, 1995b, c; Resnick and Goergen, 1995).

2.4.18 Aplasia of Ulnar Styloid Process

In some individuals, the styloid process of the distal ulna forms as a separate ossification centre (Barnes, 2012a). When this centre fails to ossify, it is termed aplasia of the styloid process (Figure 2.33).

Figure 2.33: Aplasia of the ulnar styloid process of the distal right ulna in an adult possible male. (**A**) Posterior view. (**B**) Anterior view. (CN02, Wharram Percy)

2.4.19 Thanatophoric Dysplasia

Thanatophoric dysplasia is a lethal form of short-limbed dwarfism (Donnelly *et al.*, 2010; Li *et al.*, 2005; Naveen *et al.*, 2011). Thanatophoric is from the Greek meaning death-bearing and the defect leads to death *in utero* or shortly after birth (Naveen *et al.*, 2011). It is one of the most common lethal skeletal dysplasias, occurring in 1 in 20,000 to 1 in 50,000 births from developed countries (Li *et al.*, 2005; Naveen *et al.*, 2011; Stevenson *et al.*, 2012). The dysplasia is caused by a sporadic mutation of the *FGFR3* gene affecting the formation and growth of bone and cartilage (Barbosa-Buck *et al.*, 2012; Li *et al.*, 2005; Naveen *et al.*, 2011; Stevenson *et al.*, 2012). Individuals with thanatophoric dysplasia show anterior narrowing of the skull, frontal bossing, a depressed nasal bridge, a short neck, a narrow thorax, short ribs, platyspondyly (flattened vertebral bodies), a narrow spinal canal, protruding abdomen, short and broad pubic bone and ischium, short limbs, and flared metaphyses (Cope and Dupras, 2011; Li *et al.*, 2005; Naveen *et al.*, 2011). In type 1, the femora are curved to resemble telephone receivers (curved

along the shaft with flared metaphyses at each end) and the skull is sometimes clover leaf-shaped, *i.e.* tri-lobed due to premature closure of the coronal and lambdoidal sutures (Figure 2.34) (Donnelly *et al.*, 2010; Li *et al.*, 2005). In type 2, the femora are straight and the skull is always clover leaf-shaped.

Figure 2.34: Lower limb bones of an individual with thanatophoric dysplasia showing flared metaphyses and telephone receiver shaped femora (arrows) in fœtal remains. (CS06 684, South Shields)

2.4.20 Congenital Defects Conclusions

This section has described the congenital defects observed in this study. Many of these anomalies are caused by known teratogens (Section 2.2) that may have been present in 18th and 19th century Northeast England (Section 2.3). Therefore, it may be possible to use congenital defects to distinguish different patterns of detrimental living conditions in the past. Below, "stress" indicators are discussed as they can be an additional measure of poor living conditions in the past as evidenced in human remains.

2.5 "Stress" Indicators Literature Review

'...although all of these markers are nonspecific indicators of health, when analyzed together they create a composite profile of general health and quality of life.'
(Šlaus, 2008)

Section 2.3 showed that the living environments of both the urban and rural study sites could be detrimental to health. By examining "stress" indicators, it may be possible to examine the living conditions individuals were exposed to in past populations as they are typically thought of as signs of poor health. Porotic hyperostosis, cribra orbitalia, dental enamel hypoplasia, periosteal new bone formation, and stature can all be categorised as "stress" indicators. These are various expressions of the body's physiological response to an external stressor such as malnutrition, infection, or air pollution (Martin *et al.*, 1985; Mays, 2012b; Pollard, 1999). Therefore, differences between populations in height and the frequency of "stress" indicators may show differences in living conditions. As in Section 2.4, this section details the definition and descriptions of each "stress" indicator observed in this study.

2.5.1 Porotic Hyperostosis and Cribra Orbitalia

Porotic hyperostosis and cribra orbitalia are commonly recorded non-specific "stress" indicators in bioarchaeological research. Porotic hyperostosis (PH) is described as the expansion of the diploë, between the two tables of the skull, and thinning of the outer table, creating a sieve-like appearance (Figure 2.35A) (Amadi *et al.*, 2012; Grauer, 1993; Martin *et al.*, 1985; Mnapo *et al.*, 2013; Stuart-Macadam, 1992a, b). PH on the cranial vault occurs most often symmetrically on the frontal and parietal bones but can sometimes occur on the occipital bone (Aufderheide and Rodriguéz-Martín, 1998; Stuart-Macadam, 1992a, b; Walker *et al.*, 2009). Cribra orbitalia (CO) is the term given to porotic hyperostosis localized to the roof of the orbits (Figure 2.35B) (Exner *et al.*, 2004; Obertová and Thurzo, 2008; Stuart-Macadam, 1992a; Walker *et al.*, 2009; Wapler *et al.*, 2004). CO is most commonly bilateral and appears in the anterolateral portion of the orbital roof (Aufderheide and Rodriguéz-Martín, 1998).

Figure 2.35: (**A**) Porotic hyperostosis on a parietal bone fragment in a possible female 25-29 years old, with the area of thickening of the diploë indicated by the arrow. (COL10 063, North Shields) (**B**) Cribra orbitalia of the left orbital surface in a non-adult about 12 years old. (COL10 230, North Shields)

The aetiology of these lesions is thought to be a skeletal response to anæmia with the changes observed due to increased red blood cell (RBC) production (Martin *et al.*, 1985). PH and CO were originally linked to iron-deficiency anæmia caused by diets deficient in iron, or iron loss through diarrhoea and haemorrhage due to intestinal parasites (Grauer, 1993; Obertová and Thurzo, 2008; Šlaus, 2008; Stuart-Macadam, 1992a, b; Walker *et al.*, 2009). However, more recent studies attribute the cause to other biological causes (Sullivan, 2005; Walker *et al.*, 2009; Wapler *et al.*, 2004). Walker *et al.* (2009) implicate hemolytic and megaloblastic anæmias. Hemolytic anæmia is the premature destruction of RBCs and is caused by genetic conditions (*e.g.* sickle cell anæmia, thalassemia), other diseases (*e.g.* some cancers, cyanotic heart disease), and some toxins. Megaloblastic anæmia is the lack of production of RBCs most commonly arising due to chronic dietary deficiencies or malabsorption of Vitamins B_{12} and B_9 (folic acid) caused by parasitic infections and diarrhoeal disease. Wapler *et al.* (2004) indicate that not all CO is caused by anæmia but can instead be caused by inflammation and osteoporosis. While the underlying aetiology of PH and CO is perhaps uncertain, all studies do agree that the presence of these lesions indicates the existence of stressors in the form of dietary deficiencies, disease, and parasites.

2.5.2 Dental Enamel Hypoplasia

Dental enamel hypoplasia (DEH), a defect in the structure of tooth enamel due to a stressor in early childhood, has been a commonly used indicator of nonspecific "stress" such as malnutrition and disease (Figure 2.36) (Aufderheide and Rodriguéz-Martín, 1998; Garcin *et al.*, 2010; Liebe-Harkort, 2012; Obertová and Thurzo, 2008; Ogden *et al.*, 2007; Rose *et al.*, 1985; Šlaus, 2008). The defect or thinning of enamel is the result in a suspension of ameloblastic activity due to the body diverting energy away from tooth formation in response to an insult (Aufderheide and Rodriguéz-Martín, 1998; Garcin *et al.*, 2010; Goodman, 1998; King *et al.*, 2005; Liebe-Harkort, 2012; Rose *et al.*, 1985). It represents an acute episode of several weeks to two months rather than a long-term period of "stress" (Aufderheide and Rodriguéz-Martín, 1998; Rose *et al.*, 1985). As DEH occur only during formation of the enamel, the "stress" must occur while tooth formation is taking place, generally before the age of six; DEH cannot develop in a tooth after its crown development is complete (Garcin *et al.*, 2010; King *et al.*, 2005; Ortner, 2003). DEH appear as linear horizontal grooves or pits on the tooth crown and may be seen only on the facial or exteriorly-facing surfaces of a tooth (Aufderheide and Rodriguéz-Martín, 1998; King *et al.*, 2005; Liebe-Harkort, 2012; Ogden *et al.*, 2007). If more than one line is present on a given tooth, it represents more than one period of "stress" (Aufderheide and Rodriguéz-Martín, 1998). The location of the DEH can be measured relative to the cemento-enamel junction to estimate the age at which the "stress" leading to the defect occurred, although the accuracy of this has been debated (King *et al.*, 2005).

Figure 2.36: Linear pitting form of dental enamel hypoplasia on the permanent maxillary first and second incisor and canine teeth of a non-adult 5-10 years old. (F232, Fewston)

2.5.3 Periosteal New Bone Formation

Periosteal new bone formation (PNBF) is a reaction of the periosteum (the soft-tissue membrane covering bone) to some stimulus, leading to a deposit of woven bone that is remodeled with time into lamellar bone (Figure 2.37) (Boel and Ortner, 2013; Grauer, 1993; Weston, 2008, 2012). It can occur on any bone but is most commonly reported on the long bones of the arms and legs (Weston, 2012). PNBF is an inflammatory response to damage that may be caused by infection, ulceration, hypertrophic osteoarthropathy, or trauma to adjacent soft tissues (Assis *et al.*, 2011; Boel and Ortner, 2013; Šlaus, 2008; Weston, 2008, 2012). PNBF has frequently been recorded as an indicator of nonspecific "stress" or infection, particularly when it occurs in the lower leg bones (DeWitte, 2010; Grauer, 1993; Robb *et al.*, 2001; Shuler, 2011; Šlaus, 2008; Weston, 2008, 2012). However, as Weston (2012) explains, the body's response to a "stress" is to actually prohibit bone formation, meaning the presence of PNBF cannot possibly be caused by a stressor. As PNBF is a response to an individual's living environment , this study includes it as an indicator of poor living conditions under the heading of "stress indicator" even though "stress indicator" is perhaps no longer the correct term for PNBF.

Figure 2.37: (**A**) Woven periosteal new bone formation (grey area pointed out by arrow) of the right tibia in an adult of undetermined sex. (CS06 910, South Shields) (**B**) Lamellar periosteal new bone formation of the right tibia in a female over 45 years old. (CS06 107, South Shields)

2.5.4 Stature

Stature has been frequently used as an indicator of overall health and nutrition in a population (Arcini *et al.*, 2012; Watts, 2011), as attained adult stature can be affected by genetic and environmental factors (Bielicki, 1998; Steckel and Floud, 1997). The influence of genetics on height can be through environmental factors such as poor nutrition, poor health, and pollutants (*e.g.* lead, mercury, dioxins) (Arcini *et al.*, 2012; Bielicki, 1998; Briend, 1998; Haviland, 1967; Schell, 1998; Watts, 2011). The health insults created by these environmental factors leads to the body drawing away energy and nutrients from bone growth and instead focusing it on overcoming the insult, that leads to short adult stature (Watts, 2011). Since many of the environmental factors that may affect stature are associated with lower socioeconomic status, people from a high socioeconomic status are generally taller within a region (Haviland, 1967; Steckel and Floud, 1997; Steegmann, 1998; Tainter, 1980; Ulijaszek, 1998). Some catch-up growth is possible, which means that individuals severely affected by stresses in early life may be able to attain full or nearly full height due to increased growth if their situation has improved before they have finished growing (Briend, 1998; Gilbert, 1985; Golden, 1998; Steckel and Floud, 1997; Watts, 2011).

2.5.5 "Stress" Indicators Conclusions

As this section has explained, "stress" indicators can be caused by external factors such as malnutrition, infectious disease, trauma, and pollutants. These features were found in the post-medieval period of the urban and rural sites of this study (Section 2.3) and some may have been teratogenic (Section 2.2). Therefore, these indicators, as well as congenital defects (Section 2.4), can be used to gauge the health of these populations. Individuals from environments that are detrimental to health will have higher frequencies of "stress" indicators and a shorter stature than those from cleaner, healthier areas. This chapter has shown that congenital defects and "stress" indicators theoretically could have existed and be observed in past populations. In the next section, bioarchaeological evidence for the presence of congenital defects in 18th and 19th century populations from Britain will be presented.

2.6 Bioarchaeology Literature Review

'There is a tendency for [congenital defects] to be ignored in palaeopathology for the more dramatic and more common conditions such as infectious disease.'
(Roberts and Manchester, 2005:49)

In Section 2.4 it has been shown that a wide variety of congenital defects have been reported in the modern clinical literature while Sections 2.2 and 2.3 have demonstrated that teratogenic conditions may have been responsible for the development of congenital defects in the post-medieval period. This section explores published and unpublished bioarchaeological reports for information on the presence of the congenital defects described in Section 2.4. Firstly, studies that have looked at one or more skeletal samples, either across regions or through time, will be discussed. These studies have considered skeletal samples from Europe, Siberia, and North America. These multiple population studies are important as they can show changes over time or differences between groups. Furthermore, they can suggest genetic relationships, and the effect of cultural practices or environment/living conditions on the occurrence of defects. Secondly, reports

looking at one population or an individual will also be examined. These types of report are more common than multiple population studies. They show that a particular defect was present in an individual at a specific time and location, but little else can be learned without comparisons being made with other time periods or regions. These individual reports have been restricted to the 18th and 19th centuries in Britain because this period is the main focus of the current study.

2.6.1 Multiple Populations Studies

To the author's knowledge, only five "multiple populations" studies in bioarchaeology have been undertaken to observe the frequency of congenital defects. The pioneer in this field was Ethne Barnes and her 1994 study on Native Americans, with much of her work discussing the development of defects occurring in the axial skeleton (Barnes, 1994). Additionally, a review of known archaeological examples is given, along with the information needed to diagnose the defects and a description of possible symptoms. As a test study for recording these defects archaeologically, and exploring the potential information their presence could provide, four groups of skeletons from the Pajarito Plateau region of New Mexico, USA were examined, representing the Puye, Otowi, Tsankawi, and Tsirege peoples. Among the groups, 67% of all the individuals were found to have a developmental defect of the axial skeleton, although most were very mild. Of these individuals, approximately one third exhibited more than one defect. A common gene pool was suggested to explain the similar pattern of defects amongst the groups. Additionally, since they all shared the same environment, the differences in frequency between the communities could be accounted for by slight variations in cultural practices. In her discussions, Barnes largely focuses on the genetic (intrinsic) causes of congenital defects, which means that there is less attention paid to extrinsic factors. She does acknowledge that congenital defects can develop due to extrinsic factors, but no real discussion occurred on the topic and instead, the importance of congenital defects in research on genetic relatedness is stressed. Therefore, a fuller understanding of the development of defects and their

usefulness in research may have been achieved had extrinsic factors been given more notice.

The second multiple populations study was carried out on an Iron Age semi-nomadic population from Aymyrlyg, South Siberia (Murphy, 2000). Looking at two time periods (3rd-2nd century BC and 1st century BC-2nd century AD), the research examined around 800 skeletons for signs of developmental defects and how the defects may have led to disabilities and differential treatment. The majority of the observed defects were "occult" and not likely to have been detrimental to health or the person's appearance. There were, however, individuals who would have had abnormal outward appearances or may have suffered from physical disabilities. In the Aymyrlyg population, individuals with obvious congenital defects were buried in communal tombs, alongside individuals that skeletally had no signs of physical abnormalities. Unfortunately due to incomplete excavation records, it is not possible to know if affected individuals were buried with similar grave goods as those unaffected. Murphy (2000) proposes that the presence of individuals (especially adults) with developmental anomalies within a population can allow researchers to presume information about how society viewed these people. For instance, affected individuals may have had support measures within their community that enabled them to live into adulthood. In the case of Aymyrlyg, the common burials may be indications that disabled or physically "different" people may have been 'held in similar regard to other members of society' (Murphy, 2000:74). Of course, while these individuals may seemingly have been physically included in society, it is never possible to make conclusions about the mental or emotional levels of acceptance in society. Affected individuals may have held useful roles in society but may have been ridiculed or otherwise mistreated by others (Murphy, 2000). A limitation of this study is of course that not all congenital defects or physical deformities are evidenced in the skeleton. The "normal" individuals buried with the skeletally "different" individuals may have also been physically different through soft tissue defects. This demonstrates that it can be risky discussing disability and compassion in past populations if not careful (see Dettwyler, 1991 and Wood *et al.*, 1992) but Murphy avoids these pitfalls,

presenting a well-balanced approach and interpretation to the question of disability and social inclusion within these populations.

Thirdly, congenital defects in populations from five sites were compared to explore any differences between urban and rural environments in medieval England (Sture, 2001). The sites studied were the rural sites of Raunds Furnells, Northamptonshire (mid-10th to late 12th centuries AD; 213 skeletons) and St Martin, Wharram Percy, North Yorkshire (13th to 19th centuries AD; 377 skeletons), and the urban sites of the Hospital of St James and St Mary Magdalene, Chichester (1118 to the early 18th century; 271 skeletons), St Helen-on-the-Walls, York (12th to 16th centuries AD; 385 skeletons), and the Augustinian Friary, Kingston-upon-Hull (14th century to 1539 AD; 194 skeletons). The presence of sacralization, lumbarization, spina bifida occulta, cleft neural arches, supernumerary vertebrae, absent vertebrae, spondylolysis, and spondylolisthesis (anterior slippage of the vertebral body that can be associated with spondylolysis) were found in significant numbers in individuals buried at the sites. Less commonly, fused vertebrae, Klippel-Feil syndrome, and cleft lips/palates were recorded for individuals from most sites. When comparing the sites, congenital defects were found to be more common in people buried at urban sites than rural ones. Sture (2001) believes that this was due to poorer sanitation and crowding associated with urban environments.

In the fourth report, the prevalence of congenital defects was recorded and analyzed for populations in Slovakia to look for continuity of settlement at a location (Masnicová and Beňuš, 2003). Continuity at a site may be inferred from the observation of congenital defects due to them being caused by both genetic and cultural factors (see Section 2.2.1). If a new population replaces the indigenous group, bringing with them a new cultural system and gene pool, it may be possible to see this change by examining for congenital defects over multiple generations. The two cemetery populations from Devín, Slovakia were Devín-Hrad dating to the 11th to 12th centuries AD containing 217 skeletons and Devín-Zakostolom dating to the 9th century AD containing 112 skeletons. The most common defects recorded were cleft neural arches (24% D-H, 23% D-Z), sacralization (8% D-H, 7% D-Z), spondylolysis (7% D-H, 4% D-Z), and lumbarization (2% D-H, 0% D-Z). Other

anomalies were observed sporadically within both populations and included craniosynostosis, nasal bone hypoplasia, precondylar articular facet, Stafne defect, atlas occipitalization, block vertebrae, and congenital hip dislocation. The similarity in prevalence rates between the two time periods suggests genetic continuity over time in Devín despite being located at a crossroads for two long distance trade routes. This study is limited by the assumption that the congenital defects observed were caused solely by genetics, extrinsic factors do not come into the discussion when attempting to interpret the data. Limiting the causation to only one of the many factors that lead to the development of these defects is unlikely to give a full picture of the patterns observed.

Lastly, congenital defects were observed through time across Great Britain stretching from 1050 to 1850 AD in a literature review (Kase, 2010). In total, case studies and site reports for 53 sites were considered for evidence of cleft neural arch, spondylolysis, transitional vertebra, numerical variation in vertebrae, Klippel-Feil syndrome, butterfly vertebra, hemivertebra, sacral agenesis and hypoplasia, cleft lip and palate, craniosynostosis, absent external auditory meatus, Stafne defect, supernumerary ribs, bifid rib, pectus carinatum, os acromiale, and congenital hip dislocation. The most common anomalies were os acromiale (9.19% crude prevalence rate), sacralization (6.54% CPR), the presence of six lumbar vertebrae (6.36% CPR), and cleft neural arches (6.08% CPR). When looking at congenital defects as a whole, there was a statistically higher prevalence of anomalies in the post-medieval period (c.1550-c.1850) than in the late medieval period (c.1050-c.1550). Furthermore, when the late and post-medieval periods were compared to current day clinical findings, there was a much higher prevalence in the past populations. When the sites were divided into urban or rural groupings, there proved to be a higher prevalence of congenital anomalies in the urban group than in the rural; this was similar to the conclusions found by Sture (2001). It was proposed that the predominance of defects in the post-medieval period and urban areas of the late and post-medieval periods was due to the unsanitary living conditions, overcrowding, and atmospheric pollution found in the post-medieval and urban environments. This study would likely have been improved by limiting

the regions used. A study of the defect pattern through time in one area of Britain would have allowed for a better development of living conditions and their contribution to the formation of congenital defects.

2.6.2 Single Population Reports

In recent years, a growing number of cemeteries dating to the 18th and 19th centuries have been excavated in Britain. Many are located in London but others have been studied in Scotland and various areas of southern and middle England. Many of these sites, briefly detailed in Table 2.6, have produced reports on individuals and/or single populations that include information on a variety of congenital defects.

Site	Date	Socioeconomic Status	Number of Individuals	Bibliography
All Saints Church, Laleham, Surrey	post-medieval	lower class?	5	Webb, 2008
Baptist Chapel burial ground, Littlemore, Oxford	1862-1888	lower middle class	30	McCarthy *et al.*, 2009
Chelsea Old Church, London	1712-1842	high status	198	WORD Database, 2013a
Christ Church, Spitalfields, London	1729-1852	wealthy parish	968	Fibiger and Knüsel, 2005; Molleson *et al.*, 1993; Waldron, 1993
Cross Bones, Southwark, London	1594-1853	poor/pauper	148	Brickley *et al.*, 1999; WORD Database, 2013b
Holy Trinity Parish, Coventry	1776-mid-19th century	-	33	McKinley, 1999
Isle of Ensay, Outer Hebrides, Scotland	1700-post-1800	-	182	Miles, 1989, 1994
Launceston Castle, Cornwall	1700-1775	inmates of gaol	14	Saunders, 2006
Quaker burial ground, King's Lynn, Norfolk	19th century?	middle class	34	Mahoney, 2005
Quaker burial ground, Kingston-upon-Thames	1663-1814	middle class	360	Pitre and Lovell, 2010; Start and Kirk, 1998
Quaker burial ground, St Ives, Cambridgeshire	1687-1721	-	16	Clough and Loe, 2007
St Bartholomew's, Chester	1740-1883	lower class?	14	Lewis, 2001
St Benet Sherehog, London	1666-1853	wealthy parish	230	Miles *et al.*, 2008b
St Bride's Church, Fleet Street, London	1740-1852	wealthy parish	not provided	Black and Scheuer, 1996, 1997; Knüsel and Bowman, 1996
St George's Church, Bloomsbury, London	1800-1856	upper middle class	781	Boston *et al.*, 2009
St Luke's Church, Old Street, Islington	1733-later post-medieval	working and middle class	896	Boyle *et al.*, 2005

Table 2.6: A summary of the sites included in the "single population" literature review. The studies include both individual case studies and single population studies.

Site	Date	Socioeconomic Status	Number of Individuals	Bibliography
St Martin's, Birmingham	late 18th-19th century	working and middle class	857	Brickley *et al.*, 2006b
St Marylebone Church, London	1741-19th century	wealthy parish	301	Miles *et al.*, 2008a
St Oswald Priory, Gloucester	c.1540-1855	poor	119	Rogers, 1999
St Peter's, Barton-upon-Humber, Lincolnshire	1500-1855	-	729	Waldron and Rodwell, 2007
St Peter's Collegiate Church, Wolverhampton	mid-19th century	working class	150	Adams and Colls, 2007
St Ronan's, Iona, Scotland	1600-1800	-	37	Lorimer, 1994; O'Sullivan, 1994

Table 2.6 (continued): A summary of the sites included in the "single population" literature review. The studies include both individual case studies and single population studies.

Details on the congenital defects recorded for these sites are found below, split into defect type.

A. Craniosynostosis

Scaphocephaly, the premature fusion of the sagittal suture, was the only form of craniosynostosis recorded for the time period. This was found in two males, one 36-45 years old and the other 26-35 years old, at St Marylebone, London (Miles *et al.*, 2008a).

B. Cleft Lip and Palate

The only reported occurrence of a cleft lip or palate in the 18th or 19th century comes from an adult male at St Martin's, Birmingham (Brickley *et al.*, 2006b). The cleft occurred unilaterally on the right side of the maxilla and affected both the alveolar bone, presumably underlying the affected cleft lip, and palate. This was the only example found out of 391 individuals with one or more maxilla present (prevalence of 0.26%).

C. Elongated Styloid Process

Eagle's syndrome, or elongated styloid process of the temporal bone, was seen in one male over 46 years of age at St Marylebone, London (Miles *et al.*, 2008a).

D. Cleft Neural Arch

Thirteen of the sites with human remains from the 18th and 19th century reported the presence of cleft neural arches. The sites containing at least one individual with a cleft neural arch were:

- Chelsea Old Church, London
- Christ Church, Spitalfields, London
- Cross Bones, Southwark, London
- Holy Trinity Parish, Coventry
- Isle of Ensay, Outer Hebrides, Scotland
- Quaker burial ground, St Ives, Cambridgeshire
- St Benet Sherehog, London
- St George's Church, Bloomsbury, London
- St Luke's Church, Old Street, Islington
- St Martin's, Birmingham
- St Marylebone Church, London
- St Peter's Barton-upon-Humber, Lincolnshire
- St Peter's Collegiate Church, Wolverhampton

Many of the authors of the reports examined here actually erroneously used the term spina bifida occulta (see Section 2.4.4) to label what they were observing. From descriptions or definitions given in the reports, it appeared they were actually describing cleft neural arch instead of spina bifida occulta. Any defect labeled as spina bifida occulta but could be deemed to be cleft neural arch is included here in this synopsis.

Focusing firstly on London, 49 individuals from five sites were reported to have cleft neural arch of the sacrum (Boston *et al.*, 2009; Boyle *et al.*, 2005; Brickley *et al.*, 1999; Waldron, 1993; WORD Database, 2013a). Additionally, an unknown number of individuals were affected with cleft neural arch of the sacrum at two

other sites: St Benet Sherehog and St Marylebone (Miles *et al.*, 2008a, b). Researchers at St Marylebone also reported clefting in the T1, L4, and L5 vertebrae but did not provide information on the number of individuals affected for each bone element (Miles *et al.*, 2008a). At St Luke's, Islington, cleft neural arches were found at the first cervical vertebra and twelfth thoracic vertebra in one individual each (Boyle *et al.*, 2005). The seventh cervical vertebra was found to be clefted in one individual at St George's Church, Bloomsbury (Boston *et al.*, 2009).

Heading slightly north of London, sites in Wolverhampton, Birmingham, Coventry, and St Ives, Cambridgeshire report cleft neural arches (Adams and Colls, 2007; Brickley *et al.*, 2006b; Clough and Loe, 2007; McKinley, 1999). As in London, the most commonly reported region for clefting was the sacrum, seen in 23 individuals. Twenty of these individuals were from St Martin's, Birmingham, giving a true prevalence rate (TPR) of 6.83% for the defect (Brickley *et al.*, 2006b). Also reported for St Martin's was a cleft in the thoracic region (TPR=0.03%) and one in the lumbar region (TPR=0.06%) but the report does not explain which specific bone elements were affected (*ibid.*). At Holy Trinity, Coventry, one male was found to have cleft neural arches at T11 and T12 (McKinley, 1999). Further north in Barton-upon-Humber, two males aged 25-34 years old exhibited cleft neural arches across their entire sacra at St Peter's (Waldron and Rodwell, 2007).

In Scotland, the cemetery from the Isle of Ensay contained three individuals with cleft neural arches (Miles, 1989). A 30 year old male showed clefting at L5 and S1, and possibly S2. A 25 year old female showed clefting at L5 and S1, and possibly at S3-S5, while the second female had no associated description given. In the same population, clefting was also found in five individuals at the atlas. However, the site spans from 1500 to post-1800 and the author does not explain to which time period the affected individuals belong.

E. Spondylolysis

Spondylolysis was recorded at the following sites:

- Chelsea Old Church, London
- Christ Church, Spitalfields, London
- Isle of Ensay, Outer Hebrides, Scotland
- St Benet Sherehog, London
- St Luke's Church, Old Street, London
- St Marylebone Church, London
- St Oswald Priory, Gloucester
- St Peter's, Barton-upon-Humber, Lincolnshire

London again presented the bulk of reporting for spondylolysis. From five sites, 24 or 27 vertebrae (there was a discrepancy between different authors recording the defect for individuals from the crypt of Christ Church, Spitalfields) were found to have spondylolysis. It occurred once in L3 at Christ Church, Spitalfields and once bilaterally in L6 at Chelsea Old Church (Waldron, 1993; WORD Database, 2013a). More commonly affected were L4 and L5. Spondylolysis occurred at L4 in four individuals at Chelsea Old Church and was bilateral in two males and one female, and was unilateral (left) in one male (WORD Database, 2013a). Spondylolysis of L5 was seen in either 14 or 16 individuals depending on which report was used from Spitalfields (Boyle *et al.*, 2005; Fibiger and Knüsel, 2005; Miles *et al.*, 2008a, b; Waldron, 1993; WORD Database, 2013a). Eight of the occurrences were bilateral in three males and four females, with no sex information provided for the eighth. No information on laterality or the individual's sex was given for the other vertebrae. Four of the affected L5 vertebrae came from St Marylebone and a crude prevalence rate was given as 2.5% for the defect (Miles *et al.*, 2008a). Five vertebral elements, included in the overall counts above, had spondylolysis at the cemetery of St Benet Sherehog but no information was given on the individuals or vertebrae affected (Miles *et al.*, 2008b).

Northwest of London at St Oswald's Priory, Gloucester, a number of individuals was identified as having spondylolysis (Rogers, 1999). Of the fifty-eight adults with spines, 13.7% were affected with spondylolysis in the cervical vertebrae,

22.4% in the thoracic vertebrae, and 12.0% in the lumbar vertebrae. Unfortunately, no further information is given, such as the actual number of vertebrae or individuals affected, or the sex of the individuals. In Lincolnshire, researchers at St Peter's, Barton-upon-Humber found six males and four females with spondylolysis, which affected vertebrae L3-L6, although the report did not give the number of each affected (Waldron and Rodwell, 2007).

In western Scotland, spondylolysis was reported in two individuals from the Isle of Ensay (Miles, 1989). Those affected were both 50 year old males with defects bilaterally at L4. Additionally, there was a 30 year old male described as having a 'right developmentally un-united lamina L5' which appears to be a description of spondylolysis although this individual was not grouped with the others (Miles, 1989:81).

F. Block Vertebrae/Klippel-Feil Syndrome

The reports from three sites describe the presence of block vertebrae or Klippel-Feil syndrome. Klippel-Feil syndrome was seen in two individuals out of 297 with one or more vertebrae preserved for observation at St Martin's, Birmingham (TPR 0.67%) (Brickley *et al.*, 2006b). At St Peter's, Barton-upon-Humber, the prevalence rate for this condition was given as 0.3% for the entire time span of the site (c. 950-1855 AD) (Waldron and Rodwell, 2007). All occurrences were type II (confined to the cervical vertebrae) but the number of occurrences was not provided, although at least one individual (an unsexed adult over 45 years old) from the 18th or 19th centuries was found to be affected by Klippel-Feil syndrome with fusion between the fourth and sixth vertebrae. Fusion was also seen in the thoracic vertebrae in an individual excavated at St George's Church, Bloomsbury, with fusion of the second and third thoracic vertebrae identified (Boston *et al.*, 2009).

G. Transitional Vertebrae

Transitional vertebrae were observed at 16 sites:

- Baptist Chapel burial ground, Littlemore, Oxford (occipitalization)
- Chelsea Old Church, London (lumbarization, sacralization)
- Cross Bones, Southwark, London (sacralization)
- Isle of Ensay, Outer Hebrides, Scotland (lumbarization, sacralization)
- Launceston Castle, Cornwall (lumbarization, sacralization)
- Quaker burial ground, Kingston-upon-Thames (sacralization)
- Quaker burial ground, St Ives, Cambridgeshire (sacralization)
- St Bartholomew's, Chester (sacralization)
- St Benet Sherehog, London (sacralization)
- St Bride's Church, Fleet Street, London (occipitalization, sacralization)
- St George's Church, Bloomsbury, London (sacralization)
- St Luke's Church, Old Street, Islington (occipitalization, sacralization)
- St Martin's, Birmingham (occipitalization, lumbarization, sacralization)
- St Marylebone Church, London (occipitalization, lumbarization, sacralization)
- St Peter's Collegiate Church, Wolverhampton (sacralization)
- St Ronan's, Iona, Scotland (lumbarization)

Occipitalization of the atlas was described in detail for an individual named as Mrs. Mary C who died in 1802 at the age of 37 and was interred at St Bride's, London (Black and Scheuer, 1996). The atlas was fused to the occipital bone in two locations: the left superior articular facet and the posterior tubercle of the left transverse process. There was no fusion on the right side of the bone or at the arches. The union of the two bones led to a lateral displacement and rotation of the atlas to the right side, which would probably have caused the individual to tilt her head slightly to the right. In addition to this occurrence, one individual was found to

exhibit occipitalization of the atlas at St Martin's, Birmingham with a true prevalence rate of 0.33% for those remains with an occipital bone present to observe (Brickley *et al.*, 2006b). Partial occipitalization was found in an individual at St Luke's Church, Islington where the atlas was fused to the occipital condyles through an extension of the left transverse process (Boyle *et al.*, 2005). A precondylar facet was seen in one individual from the Baptist Chapel, Oxford (McCarthy *et al.*, 2009), and occipital vertebrae were observed in two females from St Marylebone, London (Miles *et al.*, 2008a).

Four sites reported lumbarization of T12: Chelsea Old Church and Cross Bones, London, Launceston Castle, and St Martin's, Birmingham. One female at Chelsea Old Church, London was recorded as having an aplastic 12th rib, demonstrating the shift (WORD Database, 2013a). At Cross Bones, London, two males, five females, and one unsexed adult were diagnosed with lumbarization of T12 leading to a crude prevalence rate of 5.41% (WORD Database, 2013b). The T12 of a male over the age of 40 at Launceston Castle showed lumbar-like facet joints (Mays and Keepax, 2006), and four individuals were found to have a lumbarized T12 at St Martin's (Brickley *et al.*, 2006b). This anomaly had a true prevalence rate at St Martin's of 1.36% for the skeletons with a preserved T12. A slightly higher true prevalence rate was found at the same site for lumbarization of S1 (1.81% for those with an S1), occurring in five individuals. The report for the site of St Marylebone, London recorded lumbarization of S1 in two males (Miles *et al.*, 2008a). Also in London, three individuals had partial lumbarization and one had complete lumbarization of the S1 at Chelsea Old Church (WORD Database, 2013a). One female from St Ronan's, Scotland was found to have her S1 lumbarized (Lorimer, 1994), and for the Isle of Ensay, Scotland, a 35 year old female was described as having a partially lumbarized S1 (Miles, 1989). The lateral process of the right side resembled that of a lumbar vertebra and the segment was not entirely fused with the rest of the sacrum.

Nearly all of the sites surveyed recorded sacralization of L5 or L6 vertebrae. Starting again with the greater London area, there were 30 occurrences of sacralization at L5 and six at L6 in individuals from six sites (Boyle *et al.*, 2005;

Brickley *et al.*, 1999; Knüsel and Bowman, 1996; Miles *et al.*, 2008a, b; WORD Database, 2013a). The L5 and L6 vertebrae were sacralized in one individual from the cemetery of St Luke's Church, Islington (Boyle *et al.*, 2005). For individuals where sex was given for sacralization of L5, five were male and one was female (Miles *et al.*, 2008a; WORD Database, 2013a). Two occurrences of sacralization at L5 were unilateral on the left, and one was bilateral (Miles *et al.*, 2008b; WORD Database, 2013a). Three of the occurrences of sacralized L5 were found in people buried in the cemetery at Cross Bones, leading to a true prevalence rate of 6.7% for the defect (Brickley *et al.*, 1999). Sacralization of the L6 vertebra occurred in four males and was partial twice and complete once (Knüsel and Bowman, 1996; WORD Database, 2013a). No other information was provided for sacralized L6 vertebrae. In addition to the sacralized vertebral elements already reported, an unknown number of sacralized L5 vertebrae were reported for people from the Quaker burial ground at Kingston-upon-Thames (Start and Kirk, 1998) and six lumbar vertebrae were sacralized in individuals buried at the cemetery of St George's Church, Bloomsbury, but there was no information on which elements were sacralized (Boston *et al.*, 2009).

Southwest from London, partial sacralization on the left side was seen in a male over 40 years of age at Launceston Castle, Cornwall (Mays and Keepax, 2006). Heading north of London, the true prevalence rate of sacralization of the L5 or L6 was 7.40% of individuals affected at St Martin's, Birmingham, with the trait found in 23 individuals (Brickley *et al.*, 2006b). Meanwhile, sacralization was found in two males at the Quaker burial ground, St Ives (Clough and Loe, 2007). Both individuals displayed complete sacralization, with one being affected in the fifth lumbar vertebra and the other in the sixth. Two individuals at St Peter's, Wolverhampton were found to have sacralization (Adams and Colls, 2007). In one of the individuals it was the fifth lumbar vertebra that was sacralized but no information was given on the other occurrence. Moving northwest, the report for St Bartholomew's Church, Chester documented two examples (Lewis, 2001). A male over 45 years of age exhibited complete sacralization of the L5 while a female over 45 exhibited unilateral fusion on the left side of the L5.

Turning to Scotland, several individuals were found with the condition across the entire site time span (1500-post-1800 AD) of the site on the Isle of Ensay but at least one male with L5 sacralized was found dating to the 18th century (Miles, 1989). Little other information was given about the total number of individuals affected and at what time in the site's history.

H. Cervical Ribs

The presence of cervical ribs was reported at the following seven sites:

- Launceston Castle, Cornwall
- Quaker burial ground, King's Lynn, Norfolk
- Quaker burial ground, St Ives, Cambridgeshire
- St Bride's Church, Fleet Street, London
- St Martin's, Birmingham
- St Marylebone Church, London
- St Peter's Collegiate Church, Wolverhampton

At St Bride's, London, a Mrs. Mary D who died in 1848 was found to have a right cervical rib present (Black and Scheuer, 1997), and also in London, cervical ribs were found in five males at St Marylebone (Miles *et al.*, 2008a). Four of these males had what the author termed a 'bony tubercle (cervical rib)' while one apparently had a true cervical rib (Miles *et al.*, 2008a:119). Between these five individuals, there were a total of six cervical ribs, implying that only one occurrence was bilateral. These findings were from observations of 4069 ribs preserved in the population (a true prevalence rate of 0.1%). At St Martin's, Birmingham, there were four out of the 264 seventh cervical vertebrae that had a cervical rib, making a 1.52% true prevalence rate (Brickley *et al.*, 2006b). No information was provided as to how many individuals were affected or if the ribs were bilateral or unilateral. Cervical ribs were suspected in one 15-18 year old male at Launceston Castle, Cornwall (Mays and Keepax, 2006). The appearance of the left side of C7 was indicative of articulations for a cervical rib but no rib was recovered, making the authors hesitant about the diagnosis. The right side of C7 had a broadened and elongated transverse process which could be diagnosed as a rudimentary rib. A

right cervical rib was found in one male at the Quaker burial ground, King's Lynn (Mahoney, 2005), and one probable cervical rib was found in a male at the Quaker burial ground, St Ives, but no details were provided (Clough and Loe, 2007). Two individuals at St Peter's, Wolverhampton were found to have cervical ribs (Adams and Colls, 2007) of which one male had a cervical rib on the left side only of his seventh cervical vertebra. No information was provided for the other example, which occurred in a female.

I. Lumbar Ribs

Fewer lumbar ribs were reported than cervical ribs in the bioarchaeological literature. At St Marylebone, London, one male was reported to have at least one lumbar rib, although the laterality is not reported (Miles *et al.*, 2008a). Bilateral lumbar ribs were also recorded for a 25 year old female buried on the Isle of Ensay (Miles, 1989). The ribs were not recovered but the L1 vertebra exhibits distinct facets.

J. Numerical Variation in Vertebrae

A numerical variation in vertebrae was reported for nine of the sites:

- Chelsea Old Church, London (L6)
- Cross Bones, Southwark, London (T13, L6)
- Isle of Ensay, Outer Hebrides, Scotland (L6)
- Quaker burial ground, Kingston-upon-Thames (C8, T13)
- Quaker burial ground, St Ives, Cambridgeshire (L6, S6)
- St Bride's Church, Fleet Street, London (L6)
- St Luke's Church, Old Street, Islington (T13, L6)
- St Martin's, Birmingham (T13, L6)
- St Marylebone Church, London (T13, L6)

Amongst the individuals interred in the Quaker burial ground at Kingston-upon-Thames, the presence of at least one eighth cervical vertebra was noted (Start

and Kirk, 1998). Unfortunately, no information was provided as to how many individuals were affected.

The presence of a thirteenth thoracic vertebra has been reported more commonly than the presence of an eighth cervical vertebra for the 18th and 19th centuries. Six individuals at St Martin's, Birmingham had the additional vertebra with a 2% true prevalence rate for the site (Brickley *et al.*, 2006b). Meanwhile, one individual was reported as having a thirteenth vertebra at Cross Bones, London (Brickley *et al.*, 1999), and also in London, the cemetery of St Marylebone contained one male and one female, both 36-45 years old with the additional vertebra (Miles *et al.*, 2008a). At least one T13 was found with an individual buried at the Quaker burial ground but, again, no information was provided about the number of affected individuals (Start and Kirk, 1998). The presence of a thirteenth thoracic vertebra was seen in one individual at St Luke's Church, Islington (Boyle *et al.*, 2005).

An additional lumbar vertebra was reported more frequently than other supernumerary vertebrae. At Cross Bones, London, one individual had six lumbar vertebrae (Brickley *et al.*, 1999). On the Isle of Ensay, Scotland, a 25 year old female had twenty-five vertebrae (Miles, 1989). As seven were described as typically cervical and twelve were typically thoracic, it can be assumed that the additional vertebra is from the lumbar region, although the report does not specifically state this. From the crypts at St Bride's Church, London, one individual, a male 35-40 years old, was found to have an L6 present (Knüsel and Bowman, 1996). The cemetery of St Marylebone, London contained five females with an additional lumbar vertebra (Miles *et al.*, 2008a). Additionally from London, at the cemetery of Chelsea Old Church six males were found to have a sixth lumbar vertebra present (WORD Database, 2013a). Of these, one occurrence of the L6 vertebra was partially sacralized and in a second it was completely sacralized. Seventeen individuals at St Martin's, Birmingham had an L6 present (TPR 5.61%) (Brickley *et al.*, 2006b). A sixth lumbar vertebra was seen in one individual at St Luke's Church, Islington (Boyle *et al.*, 2005) while two males at the Quaker burial ground, St Ives were also found to

have a sixth lumbar vertebra present (Clough and Loe, 2007). The only record of a sixth sacral segment, occurring in a male, also comes from this cemetery.

K. Scoliosis

Individuals with scoliosis were seen at the following eight sites:

- Chelsea Old Church, London
- Cross Bones, Southwark, London
- Quaker burial ground, King's Lynn, Norfolk
- Quaker burial ground, St Ives, Cambridgeshire
- St George's Church, Bloomsbury, London
- St Luke's Church, Old Street, Islington
- St Martin's, Birmingham
- St Peter's, Barton-upon-Humber, Lincolnshire

Only reports of scoliosis that are not due to traumatic or underlying osteoporotic changes, but instead could be described as either congenital or idiopathic are included here. For London, fourteen individuals with scoliosis were reported from four locations: St George's Church, St Luke's Church, Cross Bones, and Chelsea Old Church (Boston *et al.*, 2009; Boyle *et al.*, 2005; WORD Database, 2013a, b). As scoliotic changes are never the same from individual to individual, the reported occurrences will be described. Scoliosis, presumably idiopathic, was seen in two females at St George's Church, Bloomsbury (Boston *et al.*, 2009), and several occurrences of idiopathic and congenital scoliosis were found in individuals buried at St Luke's Church, Islington (Boyle *et al.*, 2005). The first individual had scoliosis between T3 and T9, creating a 60^{o} curve to the left. These vertebrae were fused with five or six ribs fused to the vertebrae affected. The second occurrence of idiopathic scoliosis occurred in the same location of the spine (T3-T9) in another individual but no other details were given. The third individual with idiopathic scoliosis displayed a spinal curve to the right at L2 and to the left at T12, with several vertebrae being fused, and the ribs showing modification on the right side due to the curvature. The only example of congenital scoliosis in an individual was due to a cleft of the anterior body of the sacrum (S1-S3) creating a right-sided curve. The fifth lumbar vertebra was wedged, offsetting the curve. One male from

the cemetery at Cross Bones demonstrated congenital scoliosis caused by incomplete lumbarization of S1, leading it to tilt to the right (WORD Database, 2013b). Researchers at Chelsea Old Church recorded five females, one male, and one unsexed individual with idiopathic scoliosis (WORD Database, 2013a). In two of the females, a change to the ribs due to the abnormal curvature was described.

North of London, scoliosis was described in individuals from St Martin's, Birmingham and the Quaker burial grounds of King's Lynn and St Ives (Brickley *et al.*, 2006b; Clough and Loe, 2007; Mahoney, 2005). Congenital scoliosis was reported in eleven individuals (true prevalence rate of 3.32%) at St Martin's, Birmingham (Brickley *et al.*, 2006b). In one male the scoliosis was caused by multiple hemimetameres occurring at T3 through T5. No information on the other individuals was provided. At the Quaker burial ground of King's Lynn, slight scoliosis was seen in one female (Mahoney, 2005). The spine exhibited the typical "s" shape with curves to the right at T10 and to the left at T5. One male at the Quaker burial ground, St Ives displayed idiopathic scoliosis (Clough and Loe, 2007). The spine formed a double curve in the thoracic vertebrae and was fused from T4 to T6 and from T7 to L1. The upper ribs on both the right and left sides had angular deformities, several ribs were fused to vertebrae or other ribs, and many ribs were atrophied.

Further north, two females were recorded with idiopathic scoliosis from the churchyard of St Peter's, Barton-upon-Humber (Waldron and Rodwell, 2007). The first individual showed a right curve in the thoracic vertebrae, while the second had a left curve at T3-T5 and a right curve at T8-T10. The second individual also had ribs that were gracile and twisted due to the spinal curvatures.

L. Other Vertebral Anomalies

Several other vertebral anomalies were found at four locations:

- Chelsea Old Church, London
- Quaker burial ground, St Ives, Cambridgeshire
- St Luke's Church, Old Street, Islington

- St Marylebone Church, London

Several other vertebral anomalies were recorded in individuals buried at St Marylebone, London (Miles *et al.*, 2008a). A left hypoplastic transverse process was found on C7 in one 36-45 year old male, and hypoplastic superior apophyseal facets were seen on the left side of both T11 and T12 in a male over 46 years of age. Also in London, hypoplasia of the right apophyseal facets of L5 and S1 were seen in a male over 46 years old at Chelsea Old Church (WORD Database, 2013a). At St Luke's Church, Islington, one individual exhibited a hypoplastic transverse process on the second thoracic vertebra (Boyle *et al*., 2005). Hypoplasia or aplasia of various areas of the vertebrae was further recorded in individuals buried at the Quaker burial ground, St Ives (Clough and Loe, 2007). One male displayed aplasia of the left apophyseal facets on L5 and S1. This same individual also had hypoplasia of the right spinous process in the third cervical vertebra. The first cervical vertebra of one male further showed hypoplasia of the right superior apophyseal facet.

M. Bifid Ribs

Bifid ribs were found at three sites. Firstly, one bifid rib was found at St Benet Sherehog, London amongst the 85 preserved ribs giving a 1.2% true prevalence rate (Miles *et al.*, 2008b). Secondly, two occurrences of bifid rib were found at Chelsea Old Church, London, both in males (WORD Database, 2013a). Lastly, the Isle of Ensay, Scotland report provided more details as to the defect seen. One 50 years old female was affected on a left, likely, third rib (Miles, 1989). The rib was bifid at the sternal end and, because all twelve left ribs were present, this represented a bifid rib rather than a fusion of two ribs. The anterior third of the rib was approximately double the normal width and ended with two separate costochondral junctions. There was a corresponding supernumerary facet on the sternum.

N. Other Rib Anomalies

Other anomalies of the ribs were recorded at five locations:

- Quaker burial ground, King's Lynn, Norfolk
- St Luke's Church, Old Street, Islington
- St Martin's, Birmingham
- St Marylebone Church, London
- St Peter's Collegiate Church, Wolverhampton

It was reported that three individuals from the site of St Marylebone, London had fused ribs (Miles *et al.*, 2008a). Two adult males and a 1-5 year old non-adult were all affected on the first and second right ribs. In the same population, two males and five females exhibited hypoplastic twelfth ribs while one female exhibited an aplastic twelfth rib. However, the report does not state if these rib changes were bilateral or unilateral. The site report for St Luke's Church, Islington described the presence of fused ribs in one individual (Boyle *et al.*, 2005), with the shafts fused but both the costal and vertebral ends separate. Another example of rib fusion was recorded at the Quaker burial ground, King's Lynn (Mahoney, 2005), where the first and second right ribs were congenitally fused in one female. At St Martin's, Birmingham, eight individuals were affected by rib segmentation errors including broad ribs, rib spurs, bifid ribs, and fused ribs (Brickley *et al.*, 2006b). These errors occurred in 1.76% of the population. Unfortunately, the authors did not provide the number of occurrences of each anomaly separately.

O. Pectus Carinatum

The only reported occurrence of pectus carinatum comes from St Marylebone, London (Miles *et al.*, 2008a). A short, wide manubrium and sternum that exhibited a pronounced anterior curvature was found in an adult of undetermined sex.

P. Os Acromiale

Ten sites recorded the presence of os acromiale:

- All Saints Church, Laleham, Surrey
- Baptist Chapel burial ground, Littlemore, Oxford
- Chelsea Old Church, London
- Cross Bones, Southwark, London
- Isle of Ensay, Outer Hebrides, Scotland
- St Luke's Church, Old Street, Islington
- St Marylebone Church, London
- St Oswald Priory, Gloucester
- St Peter's, Barton-upon-Humber, Lincolnshire
- St Peter's Collegiate Church, Wolverhampton

In the greater London area, 17 individuals were affected with os acromiale of the scapula from the sites of Cross Bones, St Luke's Church, Chelsea Old Church, and All Saints Church (Boyle *et al.*, 2005; Brickley *et al.*, 1999; Webb, 2008; WORD Database, 2013a). Seven of the occurrences were bilateral, three were unilateral right, five were unilateral left, one was only observable for the left scapula, and the final one was only observable for the right scapula (*i.e.* both sides were not preserved well enough for observation). Additionally, 12 scapulae out of a total of 204 scapulae exhibited os acromiale at St Marylebone Church but the number of individuals affected was not given (Miles *et al.*, 2008a).

Away from London, os acromiale was observed in 37 individuals spread amongst St Oswald's Priory at Gloucester, the Baptist Chapel at Oxford, and St Peter's at Wolverhampton (Adams and Colls, 2007; McCarthy *et al.*, 2009; Rogers, 1999). The only information on laterality comes from the Baptist Chapel where the two individuals there had bilateral occurrences (McCarthy *et al.*, 2009). The affected individuals at St Peter's consisted of three females and one male (Adams and Colls 2007). Even further north, the only information provided for the presence of os acromiale was the crude prevalence rate of 3.0%, for St Peter's, Barton-upon-Humber (Waldron and Rodwell, 2007). Finally, on the Isle of Ensay, Scotland, four individuals exhibited os acromiale (Miles, 1994).

2.6.3 Bioarchaeological Literature Limitations

Several aspects were found to be lacking in surveying the bioarchaeological literature for this review. There is a scarcity of data available for the 18th and 19th centuries, the era of interest to this study. While it is becoming more frequent for post-medieval cemeteries to be excavated in Britain and the skeletal remains to be analysed, bioarchaeological data from the period still lags behind other eras. Post-medieval cemeteries are generally not excavated solely for research purposes like those from other time periods (*e.g.* Roman era cemeteries). Instead, they tend to be excavated due to modern redevelopment or emergency conservation (Cherryson *et al.*, 2012). It also appears that much of the research undertaken for this period goes unpublished (*ibid.*). Organisations such as Oxford Archaeology and the Centre for Human Bioarchaeology (Museum of London) have very useful online resources for their unpublished work, but they seem to be exceptions. Furthermore, as these organisations are located in the south of England, much of their excavation and research tends to focus on that region.

Following on from this, in general there is a paucity of bioarchaeological information on congenital defects for much of Britain. Much of the information discussed in this chapter came from London. This is most likely due to a larger number of excavations of all time periods in the capital due to demands on space for development. In London, space is at a premium so building developers may be more willing to fund the excavation and publishing of the findings from post-medieval cemeteries. Elsewhere in the country, there is not as much demand on space and therefore excavation of post-medieval cemeteries may not occur. Cherryson *et al.*'s (2012) anthology lists archaeological work in multiple post-medieval cemeteries for every county in Britain but the vast majority is unpublished. From the descriptions provided, many "excavations" were more like "watching briefs," monitoring the removal of archaeological remains by developers, of a small area of a cemetery and there was no attempt made to examine the human remains recovered.

When reports are made available, there is commonly a dearth of information about the observed congenital defects. Providing these data allows for better comparisons to be made between regional locations in order to observe for patterns of congenital defects in this time period. In general, making comparisons between a study population and contemporaneous populations in a site report is likely hindered by time constraints, but comparisons within a population can be useful and interesting. Congenital defects should be compared through time, for different socioeconomic statuses, or by sex within a cemetery to observe any patterns that may produce a better understanding of life in the 18th and 19th centuries. Another area where bioarchaeological reports could be improved is in the information provided about potential causes of observed congenital defects. Some publications have contained interesting information about the living conditions that the individuals from a given cemetery would have experienced (*e.g.* Brickley *et al.*, 1999, 2006a), but little attempt, if any, is made to draw inferences between the environmental/cultural conditions and their impact on pathological and particularly congenital changes.

2.6.4 Bioarchaeological Literature Conclusions

This section has detailed the types and frequencies of defects seen in the 18th and 19th centuries in Britain as demonstrated by published and unpublished bioarchaeological reports. Nearly all of the defects reported here were minor anomalies that would have had little impact on the lives of the individuals affected. As individuals with major congenital defects may not have been likely to survive for long without the aid of modern medicine, the presence of these minor defects can serve as a proxy for major ones (Barnes, 1994). In other words, if minor defects are found, it is likely that there were also more serious defects present in past populations too. Additionally, congenital defects in the skeleton may indicate the presence of soft tissue anomalies present within the population which cannot be seen in the vast majority of archaeological contexts. As noted in Sections 2.2, 2.3, and 2.4, the presence of congenital defects such as the ones detailed in this current

section may indicate the existence of teratogenic agents in the living environments of urban and rural contexts in the past.

2.7 Conclusion

This chapter has explored the clinical, bioarchaeological, and historical published literature to gain insight into what is known about congenital defects and living conditions in the post-medieval period. Section 2.2 showed that teratogens, environmental factors that can cause congenital defects, include factors such as fevers, malnutrition, and exposure to lead and carbon monoxide. Section 2.3 described the historical evidence for these and other potential teratogenic agents in the living environments at the four urban and rural study sites. These teratogens and poor living conditions may have led to the congenital defects and "stress" indicators that have been described in clinical and bioarchaeological literature (Sections 2.4, 2.5, and 2.6).

Having been established in this chapter that congenital defects and the poor living conditions that could have led to their formation existed in the past, the most appropriate way to acquire the data on congenital defects and "stress" indicators in past populations need to be determined. Therefore, the next chapter will detail the cemetery sites studied and their relevance to the research rationale and hypothesis. It will additionally provide the methods used to diagnose the congenital defects and "stress" indicators under investigation and the statistical analyses undertaken to determine the validity of the study hypothesis (Section 1.3).

CHAPTER 3
MATERIALS AND METHODS

'Detecting patterns of developmental defects within a...skeletal population permits interpretive projections for the occurrence of major defects, biological affinities, and both cultural and environmental influences.'
(Barnes, 1994:6)

3.1 Materials

To test the hypothesis that congenital defects were more prevalent in urban post-medieval populations, this study explores skeletal remains from cemeteries associated with two coastal urban centres and two inland rural sites in Northeast England dated largely to the 18th and 19th centuries AD (Figure 3.1). Settlements in the post-medieval period classified as urban are based largely on population size over 5,000 and an economy based on industries other than agriculture (Floud and Harris, 1997; Slack, 2000; Wrigley, 2004). Rural settlements would therefore have a smaller population and largely rely on agriculture for their main means of support, although other small-scale industries were still often practised such as stonemasonry, shoemaking, and spinning/weaving (*ibid.*). Chosen for this study were the urban sites of North and South Shields, with largely industrial based economies (Hodgson, 1903; Moffat and Rosie, 2005; Openshaw, 1978; Thornborrow, 1968, 1988; Warren, 1980). The rural sites Fewston and Wharram Percy, both in North Yorkshire with agriculture as the main employment (Baines, 1822, 1823; Parkinson, 1899; PR/WP 3), were chosen due to their geographical proximity to the urban sites.

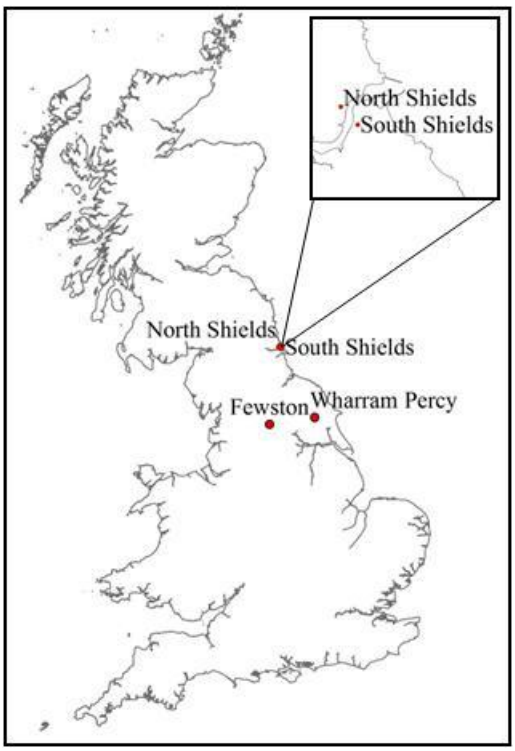

Figure 3.1: Map showing the locations of the four study sites. Contains Ordnance Survey Data ©Crown Copyright and Database Right 2013.

3.1.1 North Shields

The first urban site is that of the Quaker burial ground, Coach Lane, North Shields, North Tyneside (NZ 3533 6787) (Figure 3.2). The town of North Shields is located east of Newcastle-upon-Tyne on the north side of the mouth of the River Tyne, and is separated from the North Sea by the village of Tynemouth. The cemetery itself was located on what was at the time the northern edge of the town, approximately 250m from the riverfront. The cemetery was associated with the first Society of Friends Meeting House established in North Shields at the Bull Ring in 1698 (Alan Williams Archaeology, 2008). The burial ground was in use from 1711 until it was officially closed in 1857, although the last burial is believed to have occurred in 1841. Most members of the Friends in North Shields would have been buried here until a second Quaker burial ground was opened in 1811. Following disuse as a burial ground, the land was left as pasture until it was leased in 1907 by the North Tyneside Council for use as a public park. In 1961 three burials were discovered and removed by workmen at the site. These skeletons were later

reburied at a different location. The site was excavated in 2010 by Pre-Construct Archaeology ahead of planned redevelopment of the location.

The excavation reports describe 244 individual graves and 24 charnel features (Langthorne, unpublished-a, b; Pre-Construct Archaeology, unpublished). Preservation was largely good throughout the population with 82 individuals being >75% complete (Langthorne, unpublished-a, b). All burials were largely found in rectangular shaped earth-cut graves, with the exception of burial 72, a named individual called John Walker, who was not examined due to burial in a lead coffin, which was brick-lined with a stone slab over the top (Pre-Construct Archaeology, unpublished). A second individual, believed to be John Walker's wife Mary, was also not examined due to interment in a lead coffin. Five grave cuts contained no skeletal remains. Most graves were orientated southwest to northeast with individuals in a supine position with their heads to the southwest, although some were to the northeast. The graves were largely dug in five rows. Although against Quaker beliefs, the ground was reused leading to heavy intercutting in the northwest portion of the cemetery. Intercutting in the southeast half was less common. Burial goods were rare and mostly consisted of shroud pins. Burial occurred in coffins with their bases and sides moderately preserved in situ. The skeletal collection was examined for the purposes of a brief assessment, but no full report has been undertaken thus far. The initial report recorded 236 individual articulated skeletons (Langthorne, unpublished-a, b). However, after examining all skeletal remains for this site, it was determined by the author of this thesis that some remains labeled as charnel actually appeared to be discrete complete, or nearly complete, individuals. Consequently, this current study examines 245 individuals and the charnel remains from all 87 contexts.

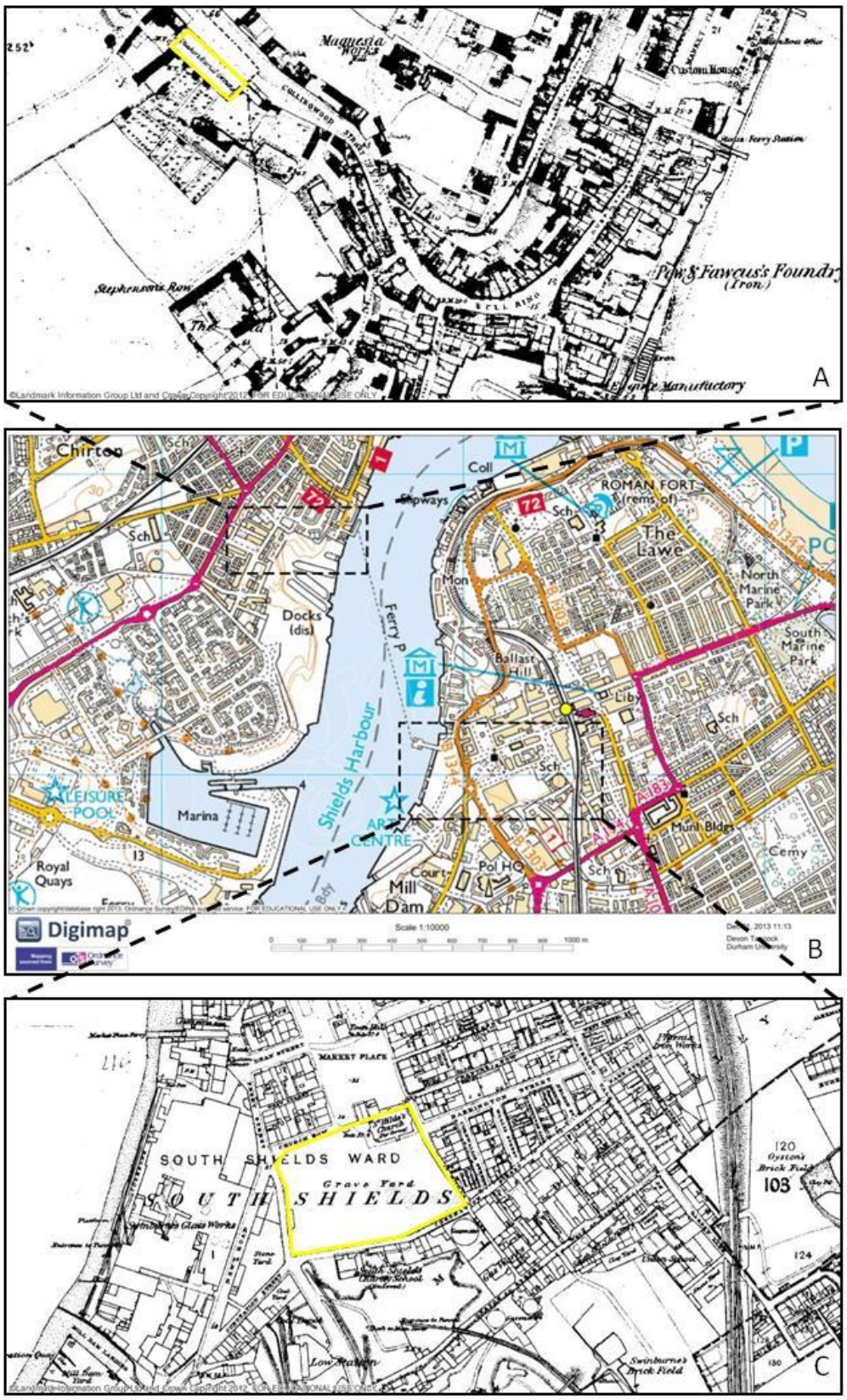

Figure 3.2: Ordnance Survey maps for the sites of North Shields and South Shields. Yellow markings show location of cemeteries in (**A**) and (**C**). (**A**) Showing 1860 map of North Shields. (**B**) Showing modern map. (**C**) Showing 1850 map of South Shields. ©Crown Copyright and Database Right 2013. An Ordnance Survey/EDINA Supplied Service.

3.1.2 South Shields

'So aw'se come back to aud St HILD's
By LIZZIE'S side to rest.'
"Epitaph on a Shields Man" by William Brockie
(Hood Coulthard, 1960:36)

The second urban site comes from St Hilda's church, Coronation Street, South Shields, South Tyneside (NZ 3615 6700) (Figure 3.2). South Shields is located east of Gateshead on the south side of the mouth of the River Tyne at the North Sea. The combined population of South Shields and nearby Westoe in 1801 was over 11,000 (Hodgson, 1903). The church and cemetery stand in the Mill Dam valley. Due to overcrowding of the cemetery, the ground was raised using ballast in 1816 providing a definitive dating point (Inkster and Speak, 1998). Burials ceased in 1856. The cemetery was excavated in 2006 due to planned redevelopment of the area. In total, 204 articulated skeletons and 50 charnel deposits were excavated (McCarthy and Clough, unpublished). Preservation was largely excellent or good but there was variation, with some skeletons being very poorly preserved. Three of the individuals had partially legible *departum* plates. Nails but no coffins were recovered from the site. All articulated and charnel remains were examined for this thesis.

3.1.3 Fewston

The first rural cemetery was the churchyard of St Michael and St Lawrence, Fewston, North Yorkshire (SE 1947 5411) (Figure 3.3). The village of Fewston is located nine miles west of Harrogate, nine miles north of Otley, and fourteen miles east of Skipton. The churchyard was in use from the medieval period until 1896 (Caffell and Holst, 2010). The skeletons analysed in this study were excavated from the north side of the church in an area of the churchyard believed to be used exclusively in the post-medieval period, although the possible presence of medieval burials could not be entirely ruled out.

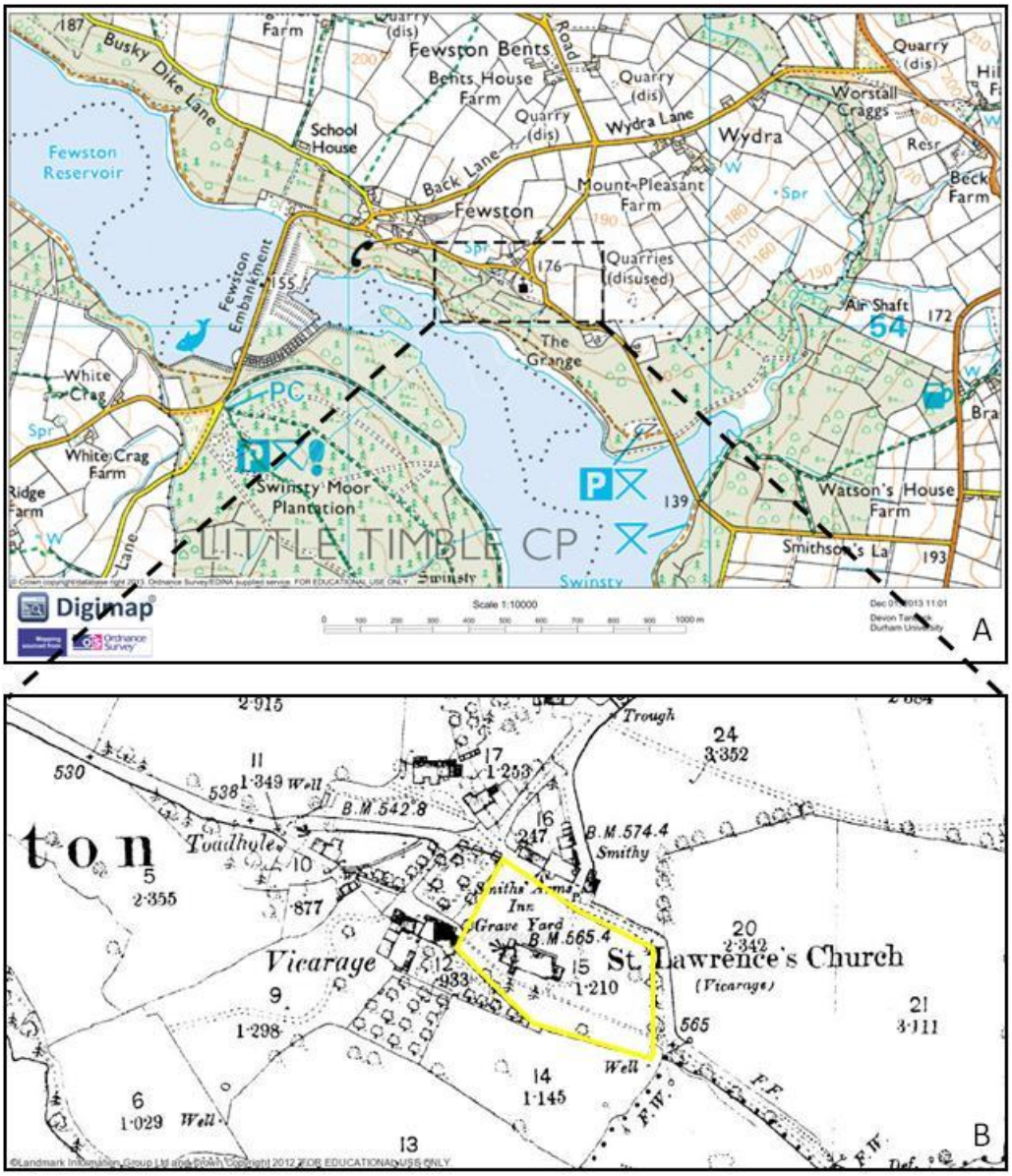

Figure 3.3: Ordnance Survey maps for the site of Fewston. Yellow marking shows location of cemetery in (**B**). (**A**) Showing modern map of Fewston. (**B**) Showing 1890 map of Fewston. ©Crown Copyright and Database Right 2013. An Ordnance Survey/EDINA Supplied Service.

The cemetery was excavated over two seasons in 2009 and 2010, with the majority of skeletons removed in the first season. All individuals were placed in an extended supine position on a west-east alignment. Fifteen individuals have been named through the presence of coffin plates, while 11 individuals are tentatively named due to their association with monuments (Caffell and Holst, 2010). A further eight individuals are possibly associated with monuments but their identification is considered questionable. After excavation, a skeletal report was compiled in which 64 individuals were fully analysed and the remaining only briefly assessed. Surface

preservation was greatly variable across the collection with only five individuals deemed good or excellent, 43 poor, and the rest classed as varying stages of well-preserved. The majority of the collection was moderately or severely fragmented and only 25 skeletons were 81-100% complete (Caffell and Holst, 2010). In total for this thesis, all 151 individuals and the charnel remains were examined from this site.

3.1.4 Wharram Percy

The second rural cemetery site is that of the cemetery associated with St Martin's church, Wharram Percy, North Yorkshire (SE 8583 6421) (Figure 3.4). The now deserted village of Wharram Percy is located in the Yorkshire Wolds about seven miles southeast of Malton. The churchyard was in use for burials from the mid-10th to mid-19th centuries, although the last burial occurred in 1906 (Beresford and Hurst, 1990; Mays, 2007b). The post-medieval burials are roughly dated to between 1540 and 1850. The village was abandoned, with the exception of one or two farms, by 1527 but St Martin's continued as the parish church for the surrounding communities of Towthorpe, Raisthorpe, and Thixendale until at least 1870 (Beresford and Hurst, 1990). Therefore, the majority of the individuals buried at Wharram Percy during the post-medieval period actually resided outside of the village.

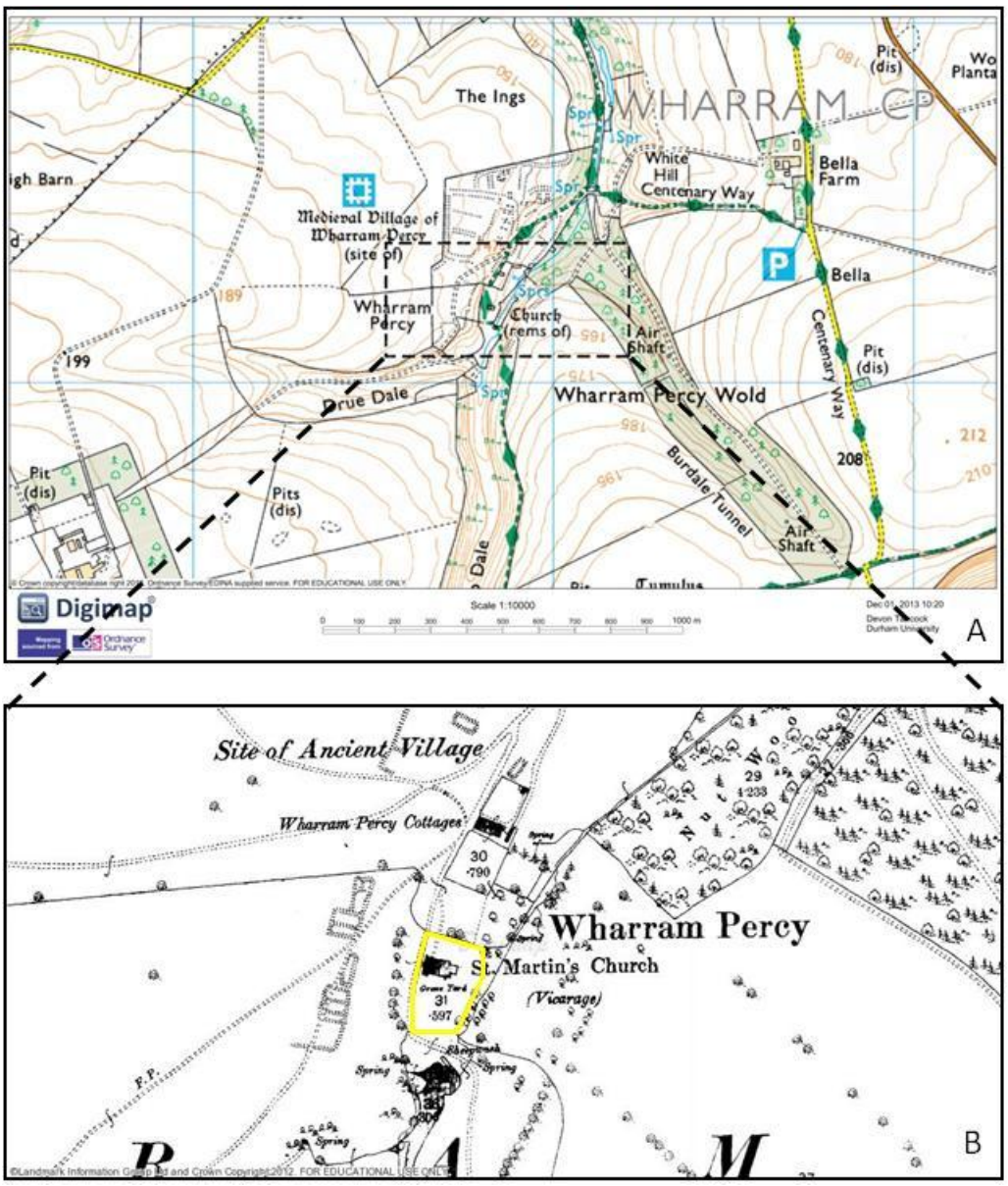

Figure 3.4: Ordnance Survey maps for the site of Wharram Percy. Yellow marking shows location of cemetery in (**B**). (**A**) Showing modern map of Wharram Percy. (**B**) Showing 1890 map of Wharram Percy. ©Crown Copyright and Database Right 2013. An Ordnance Survey/EDINA Supplied Service.

The church and cemetery were excavated over many seasons between 1962 and 1973. Over 900 skeletons were recovered from the churchyard spanning the life of use of the burial ground. Of these, 687 skeletons were fully analysed and published (Mays, 2007b). This thesis uses all 60 individuals from Wharram Percy assigned to the post-medieval period (1540-1850 AD) by Mays (2007b), based on stratigraphy, location, and, for 10 individuals, radiocarbon dating. Largely, the preservation was good and the skeletons were complete (Mays, 2007b).

3.2 Methods - Overview

All skeletons and disarticulated human remains available from the charnel deposits for all the sites and assigned to the appropriate time period were recorded for each population using forms created by the author (Appendix A contains a blank recording form). The skeleton codes for each site are as follows: COL10 for North Shields, CS06 for South Shields, F for Fewston, and CN, SA, V, and WCO for Wharram Percy. Adult skeletons were assessed for age at death using pubic symphyseal changes after Brooks and Suchey (1990), auricular surface changes after Lovejoy *et al.* (1985), and dental attrition after Brothwell (1963, 1989) as these are the standard ageing techniques used for European populations. Non-adults were aged using epiphyseal fusion data (Scheuer and Black, 2000), diaphyseal long bone length (Ubelaker, 1989), and dental development (Van Beek, 1983); again, these are generally seen as the standards used for European populations. Sex was assigned to adults based on skull and pelvic features after Bass (2005) and Phenice (1969), with pelvic features taking precedence over skull features. Using diagnostic criteria primarily laid out by Barnes (1994, 2012a) and current medical literature (see Section 2.4), the skeletons were examined for evidence of congenital defects.

Overall health of the populations was examined through the estimation of attained adult stature (Neves and Costa, 1998; Trotter, 1970) and observation of "stress" indicators (porotic hyperostosis, cribra orbitalia, enamel hypoplasia, and periosteal new bone formation of the tibia and fibula) (Goodman *et al.*, 1988; King *et al.*, 2005; Šlaus, 2008). These skeletal features can suggest an individual's or population's exposure to chronic "stress" such as poor nutrition and chronic or repeated illnesses occurring during juvenile development (Goodman *et al.*, 1988; Lewis and Roberts, 1997; Obertová and Thurzo, 2008; Šlaus, 2000; Sullivan, 2005). Their presence, and a short stature for the time period and region, might suggest the presence of poor nutrition and health, along with poor living conditions, which may have led to the development of congenital defects in individuals being born into these populations.

Whenever possible, the true prevalence rate (TPR) was calculated for the congenital defects and "stress" indicators. The TPR is the number of individuals or elements affected of the total number of individuals or elements observable for the trait. For some traits it was not possible to determine a true prevalence rate, so a crude prevalence rate (CPR) was calculated instead. The CPR is the number of individuals affected of the total number of individuals in the study regardless of the preservation of the skeletal part for observation. Whenever a prevalence rate is provided, it will be labeled as either CPR or TPR depending on the method used to analyse the data. Statistical analysis was undertaken to compare the four sites with each other and also with published data from contemporaneous sites. To identify significant differences in frequency rates of congenital abnormalities and "stress" indicators between sites, the Fisher's Exact tests were performed. Stature differences between sites were compared using a t test. Statistical significance was awarded to "p" values equal to or less than 0.05 (95%). Nearly statistically significant results were awarded to "p" values equal to or less than 0.10 (90%).

The data obtained from this study were compared to information from the following contemporaneous sites found in published literature:

- St Martin's-in-the-Bull-Ring, Birmingham (Brickley *et al.*, 2006a, b)
- St Peter's, Barton-upon-Humber (Waldron and Rodwell, 2007)
- the Cross Bones burial ground, Southwark, London (Brickley *et al.*, 1999; WORD Database, 2013b)
- Chelsea Old Church, London (WORD Database, 2013a)
- St George's Church, Bloomsbury, London (Boston *et al*., 2009)
- the Quaker burial ground, Kingston-upon-Thames (Bashford and Sibun, 2007; Start and Kirk, 1998)
- the Quaker burial ground, King's Lynn (Mahoney, 2005)
- the Quaker burial ground, St Ives, Cambridgeshire (Clough and Loe, 2007)
- the late medieval individuals from St Martin's church, Wharram Percy (Sture, 2001).

3.3 Methods - Congenital Defects

All individuals were examined for evidence of the following congenital defects:

- craniosynostosis
- cleft lip and palate
- elongated styloid process
- cleft neural arch
- hypoplastic and aplastic neural arch
- spondylolysis
- block vertebrae/Klippel-Feil syndrome
- transitional vertebra
- numerical variation in vertebrae
- scoliosis
- rudimentary and aplastic transverse process
- rudimentary apophyseal facet
- facet tropism
- supernumerary ribs
- cervical rib
- lumbar rib
- fused rib
- broad rib
- bifid rib
- pectus carinatum
- os acromiale
- aplasia of ulnar process

Additionally, one individual with unusual osteological changes was examined for thanatophoric dysplasia.

These defects were chosen due to their appearance in the bioarchaeological literature (see Section 2.6) and their likelihood of being seen bioarchaeologically. The majority of these defects are usually not so severe as to lead to premature death (particularly *in utero* or neonatally), allowing these individuals to live to late juvenile or adult ages, making their skeletons more likely to be found archaeologically. Skeletal remains of young infants and stillborns do not survive in burial contexts as well as older individuals since their bones are only partially mineralized (Lewis, 2007; Nawrocki, 1995; Saunders *et al.*, 1995; Tocheri *et al.*, 2005). Throughout history it was not uncommon for differential burial practices to apply to young or stillborn infants leading to a possible dearth of these individuals in an archaeological sample (Roberts and Manchester, 2005; Tocheri *et al.*, 2005).

These burial practices may have included an area set aside within the cemetery for these children or burial in a location outside of the cemetery (Lewis, 2007). If the entire cemetery was not excavated, the area for infants may not be uncovered. Individuals buried outside of the cemetery may not be found unless other areas of the settlement were excavated. In a specific example, burial for stillborn infants in the 17th century was undertaken by midwives in some "secret" location outside of the churchyard (Bates, 2005; Gittings, 1984). Additionally, excavation by inexperienced excavators may lead to the small bones of infants being overlooked or classified as non-human (Lewis, 2007; Tocheri *et al.*, 2005). Due to all of these circumstances, severe defects that could lead to death are unlikely to be seen archaeologically since very young individuals are not seen as commonly in archaeological collections.

3.3.1 Craniosynostosis

Craniosynostosis was diagnosed in two ways, based on the age at death of the individual. Cranial bones and fragments were examined for the presence of sections of fused sutures in non-adults too young for their cranial bones to have fused. In individuals with a fused cranium, the overall shape was examined. Those with an abnormal shape that could not be attributed to post-mortem soil compression and a missing or obliterated suture were diagnosed with craniosynostosis. Crania with an abnormal shape without any obvious involvement of prematurely fusing sutures were diagnosed as asymmetrical crania. Obliteration or absence of any portion of a suture line was recorded for each affected individual. As sutures in adults often obliterate/disappear with age and show some variation in closure timing due to sex (Key *et al.*, 1994; Meindl and Lovejoy, 1985; Milner and Boldsen, 2012), the absence of a suture or part of a suture without evidence of abnormal cranial shape was not diagnosed as craniosynostosis. In individuals diagnosed with craniosynostosis, the mandible was also examined for any changes seen and a description made.

3.3.2 Cleft Lip and Palate

Cleft lip and palate diagnosis was made through the examination of the maxilla of each individual. The presence of a cleft was recorded for any individual with a gap or notch occurring in the areas affected by these defects. Trauma and post-mortem damage were ruled out before making the diagnosis.

3.3.3 Elongated Styloid Process

An elongated styloid process was diagnosed if one or both of the styloid processes in an individual was longer than 3 cm. The process was measured using sliding calipers from the base where it joins to the temporal bone to the inferior most tip. Evidence of post-mortem breakage of the process was noted. The contour and angle of the process was recorded in order to determine if it represented an elongated bony element or an ossified ligament. The true prevalence rate was not reported. The rate of post-mortem breakage to the styloid process made the recording of observable processes difficult (*i.e.* how long does the process have to be, with or without breakage, to be classed as observable?), but only processes deemed to be elongated were recorded as present for observation.

3.3.4 Cleft Neural Arch

Unless there were obvious signs to denote spina bifida (*i.e.* broad spinal canal, flaring neural arches), all developmental gaps in the neural arch were diagnosed as a cleft neural arch. The cleft edges were rounded and did not show signs of breakage that could be determined to be postmortem damage. Signs of hypoplasia or aplasia of the structures of the arch were recorded along with the presence of the cleft. Clefting at the first, fourth, or fifth sacral arch was not classified as a developmental problem. A cleft at these locations is so common as to be considered a normal variant rather than a developmental or congenital defect (Barnes, 1994, 2012a; Conner and Ferguson-Smith, 1997; Mulhern and Wilczak, 2012).

3.3.5 Hypoplastic and Aplastic Lamina

Aplastic laminae were diagnosed by the absence of one or both sides of the neural arch in any vertebral segment. Care was taken to rule out trauma or postmortem breakages as the cause of the absent lamina(e). These anomalies were recorded as both aplastic and cleft neural arches, but counted as one defect in the overall recording of congenital defects. Unilateral hypoplastic laminae without clefting were recorded for vertebrae showing an asymmetrical appearance. Bilateral hypoplastic laminae were recorded for vertebrae with a noticeably gracile appearance. Vertebrae with clefting caused by hypoplasia of the laminae were recorded solely as having cleft neural arches, and information on the type of hypoplasia, if observable, was included in the description of the affected vertebrae.

3.3.6 Spondylolysis

Spondylolysis was diagnosed if one or more vertebra displayed a separation either unilaterally or bilaterally at the pars interarticularis. The edges of the separation on both the pars interarticularis and the neural arch (if present) were examined for evidence of post-mortem breakage. The areas of articulation between the two halves of the vertebra were examined for smooth and/or pitted surfaces. The presence of the separated neural arch was recorded, as was the laterality of the separation.

3.3.7 Block Vertebrae/Klippel-Feil Syndrome

The fusion in block vertebrae generally affects the vertebral bodies but importantly, the posterior portions are also involved (Pany and Teschler-Nicola, 2007; Resnick, 1995b). Congenital fusion can be differentiated from acquired ankylosis and fusion from diseases such as joint disease by the obliteration of the intervertebral disc space in the former. This leads to a constricted appearance at the intervertebral disc area and a trapezoidal shape to the bodies (*ibid*.). Additionally, the spinous processes are generally fused to each other in congenital occurrences but not in acquired occurrences. Therefore, block vertebrae or Klippel-Feil syndrome, were diagnosed based on an atrophic or obliterated intervertebral

disc space, trapezoidal body shape, and fused posterior elements occurring in two or more vertebral elements at any location in the pre-sacral spine.

3.3.8 Transitional Vertebra

A. Occipitocervical Border

An occipital vertebra was diagnosed based on the appearance of the base of the cranium. An expansion of the area around the foramen magnum was diagnosed as an occipital vertebra. Occipitalization of the first cervical vertebra was also diagnosed based on the appearance of the base of the cranium. Part or the entire first cervical vertebra had to be attached to the occipital bone. It can be difficult to differentiate between an occipital vertebra and occipitalization. To distinguish between the two, for the presence of an occipital vertebra the first cervical vertebra was still present and separate from the occipital bone and for occipitalization the transverse foramina were retained.

B. Cervicothoracic Border

The seventh cervical vertebra was examined for unusual morphology of the transverse processes. Cervical ribs were identified using the classification system of Barnes (1994, 2012a). Additionally, first thoracic ribs were examined for signs of facets arising from its articulation with a cervical rib, especially in skeletons lacking the seventh cervical vertebra.

C. Thoracolumbar Border

Rotation of one or both superior apophyseal facets to face medial was the diagnostic feature of lumbarization of the twelfth vertebra. The costal facet area of the vertebra was examined for signs of aplasia or hypoplasia of the costal facets. The ribs were examined for any evidence of hypoplasia. Any changes in the eleventh cervical vertebra corresponding to those seen in the twelfth were also described.

Lumbar ribs were diagnosed by the presence of costal facets on a lumbar vertebra whether an associated lumbar rib had been recovered or not. The lumbar rib, if present, was distinguished from thoracic ribs by the rounded blunt end. Thoracic ribs have a more slender, tapered appearance and are generally shorter.

D. Lumbosacral Border

Diagnosis of lumbarization involved observing the first sacral segment and the remaining sacrum. In complete sacralization, the sacrum contained only four segments and the alae of the second sacral segment were higher than the body. Incomplete sacralization had the appearance of a partially detached first sacral segment with the alae taking on the appearance of lumbar transverse processes.

Sacralization was diagnosed based on the appearance of the lowest lumbar vertebra, either the fifth or sixth. Complete sacralization was diagnosed for individuals where the lowest lumbar vertebra was completely incorporated into the sacrum. Incomplete sacralization was diagnosed when only one side of the lumbar vertebra had been incorporated. Partial sacralization was diagnosed for individuals whose lowest lumbar vertebra transverse processes articulated with the alae of the sacrum.

3.3.9 Numerical Variation in Vertebrae

In archaeological skeletal remains, it can be difficult to accurately determine the addition or subtraction of a vertebra in life. While a missing vertebra may be congenitally absent, it may just as likely be missing due to poor preservation or careless recovery (Barnes, 1994). In order to suggest that a vertebra was missing, all vertebrae present had to fit together with no potential gaps. To definitively diagnosis this anomaly, all other vertebrae had to be present in order to rule out a lack of vertebrae due to border shifting. Similarly, an additional vertebra was suggested if there appeared to be an extra vertebra in a particular spinal region. It could only be definitively diagnosed if all other vertebrae were present, resulting in

an overall pre-sacral count of 25 or more vertebral elements, or when looking at changes in the sacrum, 30 or more elements.

3.3.10 Scoliosis

Skeletal changes due to scoliosis could be seen in the vertebral body, articular facets, and pedicles as well as in the ribs (Mann and Hunt, 2005; Wever *et al.*, 1999). When placed in articulation, a lateral curve and/or "s" curve is evident. Signs of scoliosis on the vertebral body are a twisted appearance and asymmetry. The top half of the body appears to be shifted in an opposite lateral direction to the bottom half. Additionally, one side of the body appears compressed in relation to the other side. The superior and inferior articular facets are asymmetrical; one side is much larger than the other. In the instance of "s" curves, this pattern would reverse as the direction of the curve changed (*i.e.* articular facet on the right side is smaller when the spine is curved to the right, then smaller on the left side when the spine curves to the left). The pedicle thickness is also asymmetrical; one side is of a normal thickness and the other is nearly "paper thin". As for the articular facets, the pattern switches as the direction of the curve changes into an "s" shaped curve.

Scoliosis was diagnosed if one or more vertebrae exhibited more than one of these described changes due to an abnormal lateral curve (*N.B.* Diagnosis of scoliosis in a single vertebra can be tentative at best. The more complete the spine and the more changes present, the more convincing the diagnosis. In the absence of complete or nearly complete spines, changes that may have been attributable to scoliosis were recorded as potential occurrences.). If the curvature was secondary to a congenital defect of one or more vertebrae, the scoliosis was classified as congenital. In the absence of any congenital defects, the scoliosis was classified as idiopathic. Sub-classification of idiopathic scoliosis is not possible with skeletal archaeological remains as there is no way to assess how long standing the curvature was. As such, no attempt at sub-classification was made in any observed instances of idiopathic scoliosis. To attempt to determine a true prevalence rate, affected individuals were compared to individuals with at least one thoracic vertebra present for observation. While basing a diagnosis of scoliosis on one

vertebral element is not likely accurate, it could at least be recorded as potential or tentative evidence.

As scoliosis can affect areas of the body outside of the spine, other regions were investigated for changes that could be attributed to this defect. Changes can be seen in the ribs if the curve occurs in the thoracic region. Ribs on the convex side of the curve exhibit a sharper than normal angle, and those on the concave side display a wider than normal angle. The shoulder girdle, pelvic, and leg bones were also examined for any possible changes that could be attributed to scoliosis.

3.3.11 Rudimentary and Aplastic Transverse Process

A rudimentary transverse process was diagnosed based on its size. If a transverse process was visibly smaller than the one on the other side of the same vertebra or on adjacent vertebrae, it was classified as rudimentary. An aplastic transverse process was diagnosed based on the absence of the process. The affected area was examined closely to rule out trauma or postmortem damage as the cause of the absence. Laterality was recorded for both conditions.

3.3.12 Rudimentary and Aplastic Apophyseal Facet

A rudimentary apophyseal facet was diagnosed based on the size of the facet. If the facet on one vertebra was significantly smaller than the opposite side or facets on neighbouring vertebrae, it was recorded as rudimentary. An aplastic apophyseal facet was recorded when the facet was absent from the vertebral element without any signs of trauma or post-mortem breakage. Descriptions were made of complimentary facets if they were affected by the presence of either defect in the adjacent vertebra.

3.3.13 Facet Tropism

Facet tropism was diagnosed on the basis of the orientation of the apophyseal facets. While the clinical definition states that only one side of the vertebra is affected, for this study vertebrae with apophyseal facets rotated into an

abnormal orientation were recorded whether they were unilateral or bilateral. Unilateral instances were termed “facet tropism” while the bilateral instances were termed “rotated facets” so as not to deviate from the medical definition of the term facet tropism. To be diagnosed as a tropism, the facet must have been rotated to the point that it was possible to visibly detect a change, typically about 45^{o} from normal, with a full rotation being up to about 90^{o}. Rotation occurring at the facets on T11, T12, T13, or L1 was not recorded as facet tropism/rotation as these are signs of transitional vertebrae (see Section 2.4.8). These vertebrae were therefore diagnosed as transitional, but information on facet tropism/rotation was recorded in the description of the affected vertebra.

3.3.14 Supernumerary Ribs

Supernumerary ribs were diagnosed through the counting of ribs and/or vertebral segments. Thirteen or more ribs per side indicated a supernumerary rib. Extra care was taken with fragmented ribs, counting only the minimum number of ribs for each side for individuals whose ribs could not be reconstructed (*e.g.* the maximum number of heads, sternal ends, or angles present represented the minimum number of ribs present). The thirteenth rib was only counted as supernumerary if a thirteenth thoracic vertebra was present with a corresponding costal facet. Otherwise, it was not possible to confirm that a thirteenth rib was a true thoracic supernumerary rib rather than a cervical or lumbar rib. Conversely, the presence of thirteen thoracic segments, all with costal facets, indicated a supernumerary rib even if thirteen ribs per side could not be counted.

3.3.15 Other Rib Anomalies

A fused rib was diagnosed based on a merging of two adjoining ribs. A broad rib was diagnosed based on the width of the rib: roughly at least double that of surrounding ribs from the same individual. To distinguish between fused and broad ribs, the presence of one or two heads was noted. Two heads were present in fused ribs but only one in broad ribs. A bridged rib was diagnosed based on the fusion of two ribs by a narrow osseous bridge. This was differentiated from fused ribs based

on the small area of the two ribs that were joined. A bifid or bifurcated rib was diagnosed based on forking of the sternal end. The two ends were roughly the same length and have the typical sternal end appearance of any normal rib (Martin, 1960). A rib spur was diagnosed based on the presence of a bony spike or spur protruding out from the rib. This was differentiated from a bifid rib based on length and appearance of the outgrowth. A rib spur did not have a typical end appearance for a rib, rather it was rounded or sharp. It did not reach to the end of the rest of the rib and was instead less than one-third the length of the other branch (Martin, 1960). An attempt at determining true prevalence rates, or as near as true as can be expected from fragmentary skeletal remains, was made by comparing the number of affected individuals with individuals who had at least one rib present because very few individuals had completely intact ribs in this study population.

3.3.16 Pectus Carinatum

Pectus carinatum was diagnosed based on the curvature of the sternum. The sternum must have been more greatly bowed anteriorly when compared to the other skeletons examined. Partial sterna were unlikely to be diagnosed with pectus carinatum as much of the bone needs to be present to observe for the angle of bowing.

3.3.17 Os Acromiale

Os acromiale was diagnosed based on the appearance of the acromion and the portion of the acromion beyond the acromial angle needed to be missing. The edge of the acromion showed a smooth or articulating edge rather than appearing broken postmortem, and the surface of the created “joint” was porous (Figure 3.5). The presence of the separate acromion did not need to be present to lead to a diagnosis but its presence or absence was recorded.

Figure 3.5: The "joint" surface between the acromion and the os acromiale showing a porous appearance in a female over 45 years old. (CS06 107, South Shields)

3.3.18 Aplasia of Ulnar Styloid Process

Aplasia of the ulnar styloid process was diagnosed when the process was absent from the distal end of the ulna. To be diagnosed as congenital, no signs of trauma (*i.e.* avulsion fracture) were present.

3.3.19 Thanatophoric Dysplasia

Thanatophoric dysplasia was diagnosed based on the unusual morphology of the bones. Long bones were abnormally shortened with flared metaphyses, ribs were shortened, vertebral bodies displayed platyspondyly, and the pubis and ischium were shortened and broad. Individuals with bowed femora were diagnosed as having type 1, while individuals with straight femora and a cloverleaf-shaped skull were diagnosed as having type 2.

3.4 Methods - "Stress" Indicators

All individuals were examined for evidence of the following "stress" indicators: porotic hyperostosis (PH), cribra orbitalia (CO), dental enamel hypoplasia (DEH), and periosteal new bone formation (PNBF). Additionally, for sexed adults with at least one intact long bone, stature was estimated. These indicators were

chosen as they commonly occur in skeletal collections and leave traces on bone that are relatively easy to diagnose (Mays, 2012b; Šlaus, 2008). For this study, the "stress" indicators were recorded in order to evaluate the living conditions at each site, a method commonly seen in bioarchaeological literature (*e.g.* Garcin *et al.*, 2010; Lewis, 2002; Palubeckaitė *et al.*, 2002; Peck, 2013; Pētersone-Gordina *et al.*, 2013; Šlaus, 2008; Watts, 2011). These traits can be used as measurements of "stress", or external stimuli such as malnutrition, infectious disease, or air pollution, which evokes a physiological response (Martin *et al.*, 1985; Mays, 2012b; Pollard, 1999). Therefore, an increased frequency of "stress" indicators or decreased adult stature could indicate poor living conditions amongst a population.

3.4.1 Porotic Hyperostosis and Cribra Orbitalia

All crania and cranial fragments were examined for porotic hyperostosis (PH). PH was diagnosed based on the presence of a sieve-like or pinprick appearance and a thickening of the affected area. The size of the lesion and the bone affected were recorded. Cribra orbitalia (CO) was similarly diagnosed based on a sieve-like appearance. Laterality was recorded. No effort was made to grade the lesions as active or healed as this thesis is only concerned with determining that stressors existed rather than when these stressors occurred in an individual's life.

3.4.2 Dental Enamel Hypoplasia

A dental enamel hypoplasia (DEH) was diagnosed based on the presence of an enamel defect in the form of a line, groove, or linear pitting on any tooth crown in the deciduous or permanent dentition. Each tooth was recorded for the absence or presence of at least one defect, but the number of defects per tooth was not recorded. No effort was made to record the appearance or severity of the DEH except in a few individuals with extensive cuspal enamel hypoplasia (see Ogden *et al.*, 2007). Additionally, no measurements were taken to use in ascertaining the age of onset of any defects as the timing of occurrence was not important to this thesis but rather that a stressor existed at a particular site.

3.4.3 Periosteal New Bone Formation

Periosteal new bone formation (PNBF) was recorded only on the tibiae and fibulae. The tibia was selected since it is the bone most commonly affected with these lesions, and the fibula was selected since the tibial inflammatory action often spreads to its neighbouring bone (DeWitte, 2010). At least one half of the bone in question had to be present for it to be examined for PNBF. The type of new bone formation was recorded as woven, lamellar, or a mixture of the two in an effort to make this data more comparable to data gained in other studies. Also recorded were the location of the PNBF and the percentage of the bone that was affected.

3.4.4 Stature

Stature for adults was ascertained using the Trotter method (1970) as this is the method most commonly used for European populations. Only intact long bones from adult individuals that could be sexed were measured to the nearest millimeter using an osteometric board. Different bones used in this method have different error rates. In an effort to minimise there inherent errors, for males the combined femur and tibia lengths were used preferentially, followed by the femur, fibula, tibia, humerus, radius, and then the ulna. Similarly in females, the combined femur and tibia lengths were used preferentially, followed by the fibula, tibia, femur, radius, ulna, and then the humerus. Bones from the right side were preferentially chosen unless a bone from the left side was a bone that provided a lower error rate.

3.5 Conclusions

Four study sites from both urban and rural locations were examined for a wide variety of congenital defects and “stress” indicators. The congenital defects made up the bulk of this bioarchaeological research. Section 3.3 detailed the defects being observed and how they were diagnosed for the purposes of this study. These congenital defects were examined to gain further insight into the

impact of the industrial revolution on populations living in Northeast England. Meanwhile, the “stress” indicators detailed in Section 3.4 were chosen to serve as proxies, to demonstrate the presence or absence of poor living conditions at each site. In the next chapter, the results generated through the analysis of the skeletal remains will be provided.

CHAPTER 4
RESULTS

'...some anomalies appear to be common while others are very rare yet important even if only one or a few individuals are affected.'
(Barnes, 2008:361)

4.1 Introduction

This chapter details the results generated from the bioarchaeological analysis of all skeletal remains from the four study sites located at North Shields (NS), South Shields (SS), Fewston (Fe), and Wharram Percy (WP) in an effort to investigate the study hypothesis (Section 1.3). The methods used to diagnose the presence of congenital defects and "stress" indicators have already been described in Chapter 3. This chapter first details the demographic profile of the people buried at each site, looking at age and sex within each population. Next, the congenital defects seen in the study populations are described and statistical analyses provided where appropriate. Finally, the evidence for "stress" indicators is also detailed and statistical analyses provided.

Supplementary material to this chapter can be found in the Appendices B through E. Information included there contains data on age, sex, stature, the presence of "stress" indicators, and the presence of congenital defects for each skeleton recorded in this study.

4.2 Demography

4.2.1 North Shields

The Quaker burial ground, Coach Lane, North Shields contained 245 discrete individuals and 87 charnel contexts dating from 1711 to 1857. Of the 245

individuals examined, 162 were adults (66.1%) while 83 were deemed to be non-adults (33.9%). All adults were assessed for sex with 46 males, 35 possible males (50.0% M/?M), 15 females, 48 possible females (38.9% F/?F), and 18 for whom sex could not be determined (11.1% U) (Figure 4.1). The age distributions for both the adults and non-adults can be found in Figures 4.2 and 4.3.

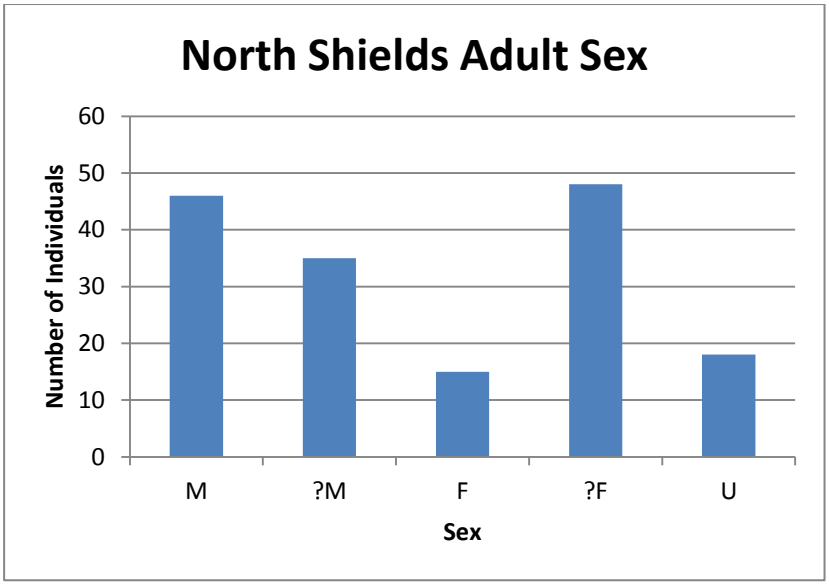

Figure 4.1: Sex breakdown of all adults from the Quaker burial ground, Coach Lane, North Shields. M = male; ?M = possible male; F = female; ?F = possible female; U = sex was undeterminable

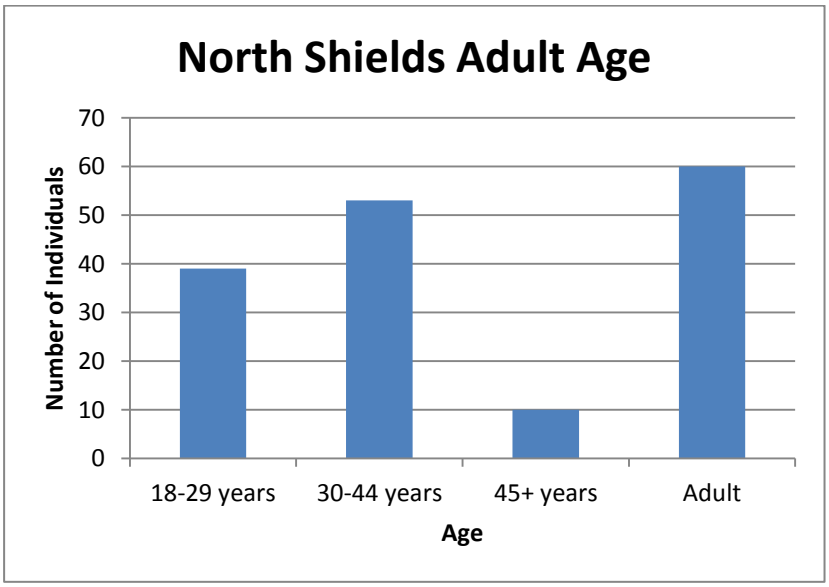

Figure 4.2: Age at death groups of the Quaker burial ground, Coach Lane, North Shields adults. Adult denotes that no age other than 18+ years old could be determined.

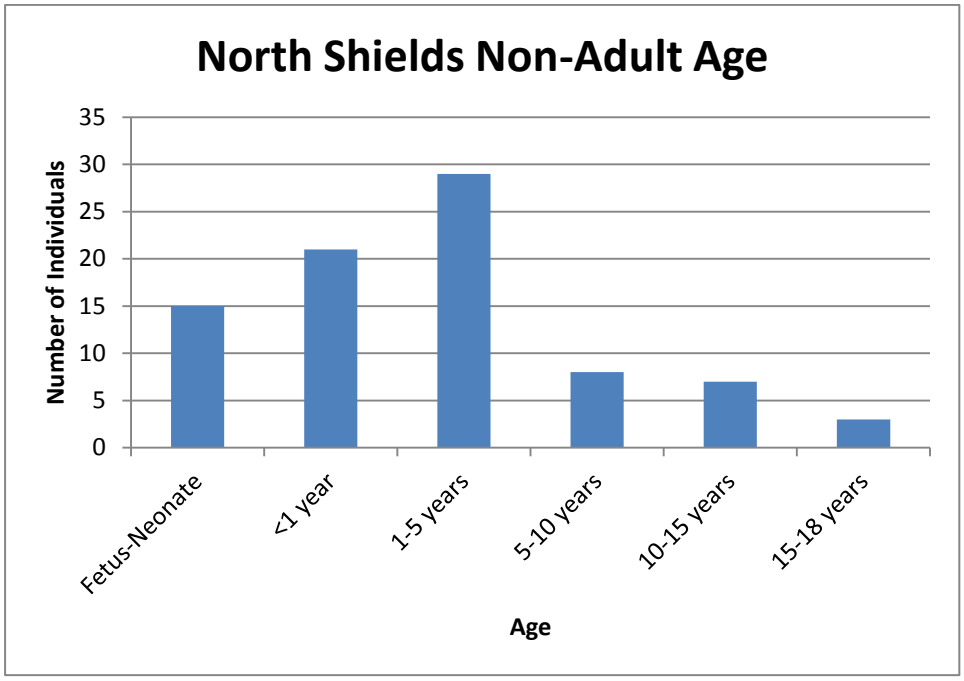

Figure 4.3: Age at death groups of the Quaker burial ground, Coach Lane, North Shields non-adults.

4.2.2 South Shields

Upon excavation, the cemetery at St Hilda's Church, Coronation Street, South Shields was found to contain 204 discrete individuals and 50 charnel deposits dating from 1816 to 1856. Of the 128 adults, 43 were assessed as male, 16 as possibly male (46.1% M/?M), 28 as female, 16 as possibly female (34.4% F/?F), and the remaining 25 could not be sexed (19.5% U) (Figure 4.4). Out of the total population, 62.7% were adults (128/204) and 37.3% were non-adults (76/204). The age groups for all adults and non-adults can be found in Figures 4.5 and 4.6.

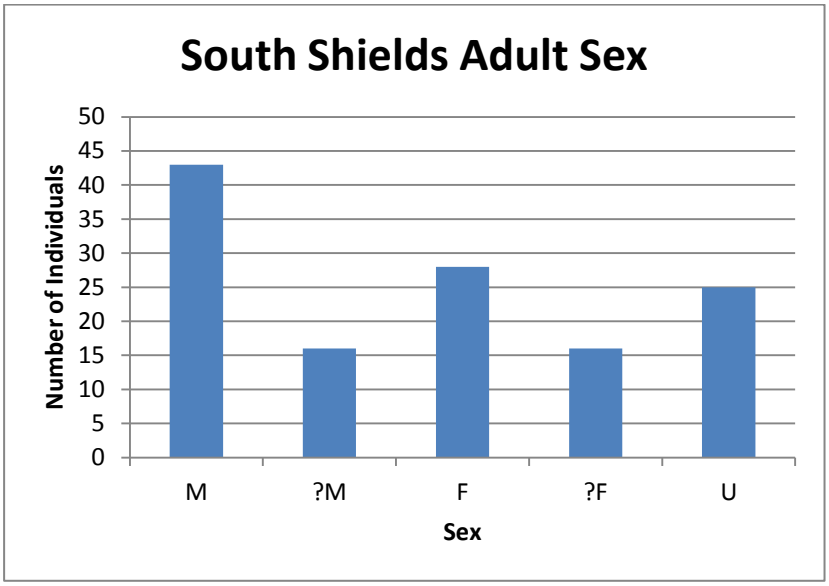

Figure 4.4: Sex breakdown of all adults from St Hilda's Church, Coronation Street, South Shields. M = male; ?M = possible male; F = female; ?F = possible female; U = sex was undeterminable

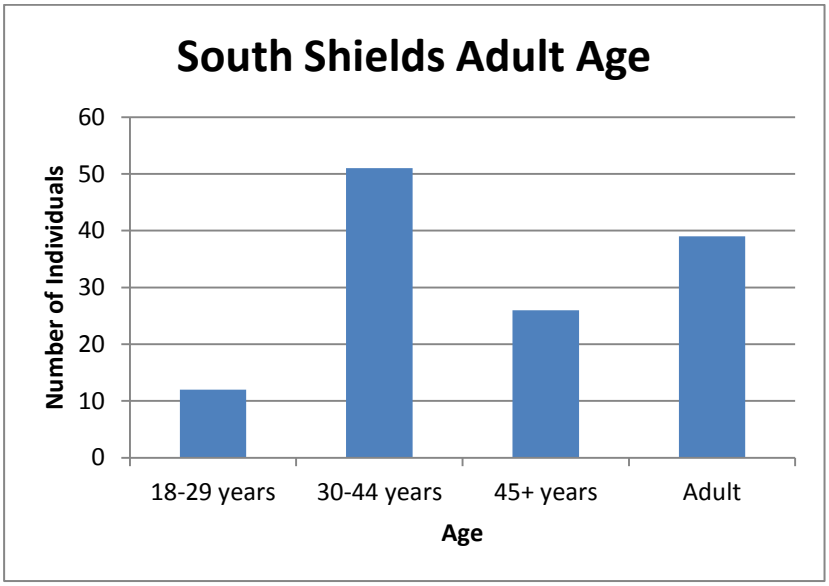

Figure 4.5: Age at death groups of the St Hilda's Church, Coronation Street, South Shields adults. Adult denotes that no age other than 18+ years old could be determined.

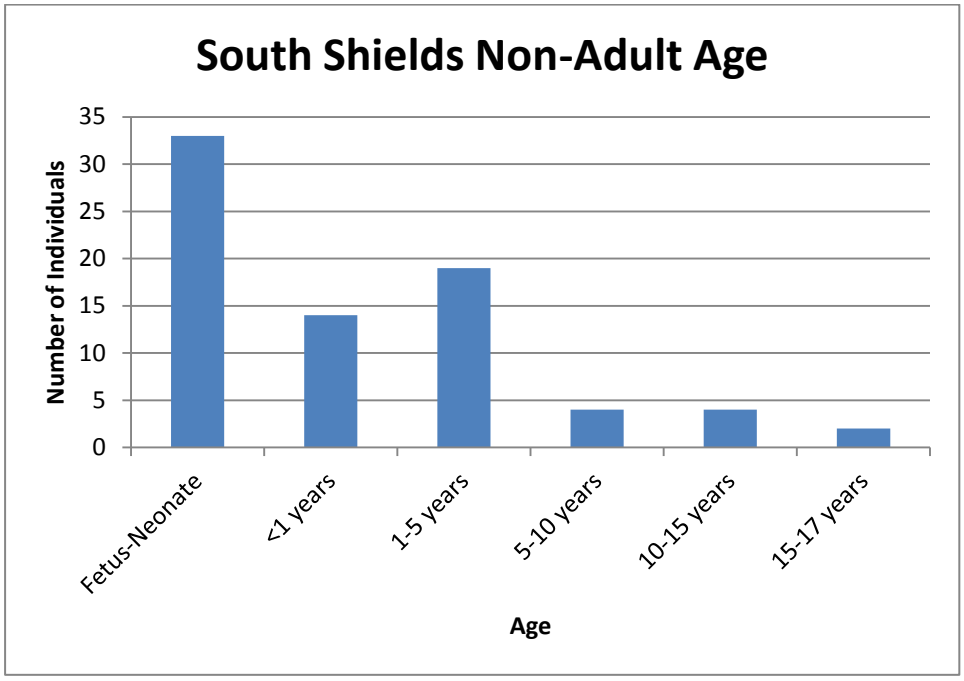

Figure 4.6: Age at death groups of the St Hilda's Church, Coronation Street, South Shields non-adults.

4.2.3 Fewston

One hundred and fifty-one individuals as well as charnel deposits were excavated from the churchyard of St Michael and St Lawrence, Fewston dated to the post-medieval period with the last burial from 1896. Of the 151 individuals, 104 were adults (68.9%) and 47 were non-adults (31.1%). The sex distribution at the site consisted of 17 males, 17 possible males (32.7% M/?M), 15 females, 28 possible females (41.3% F/?F), and 27 undetermined (26.0 % U) (Figure 4.7). Figures 4.8 and 4.9 show the age distributions for both the adults and the non-adults. Additionally, charnel deposits were examined.

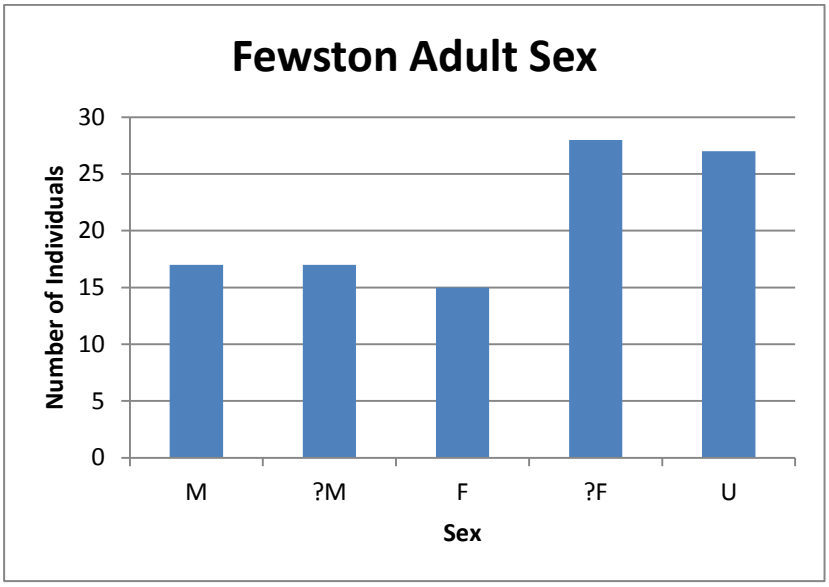

Figure 4.7: Sex breakdown of all adults from the church of St Michael and St Lawrence, Fewston. M = male; ?M = possible male; F = female; ?F = possible female; U = sex was undeterminable

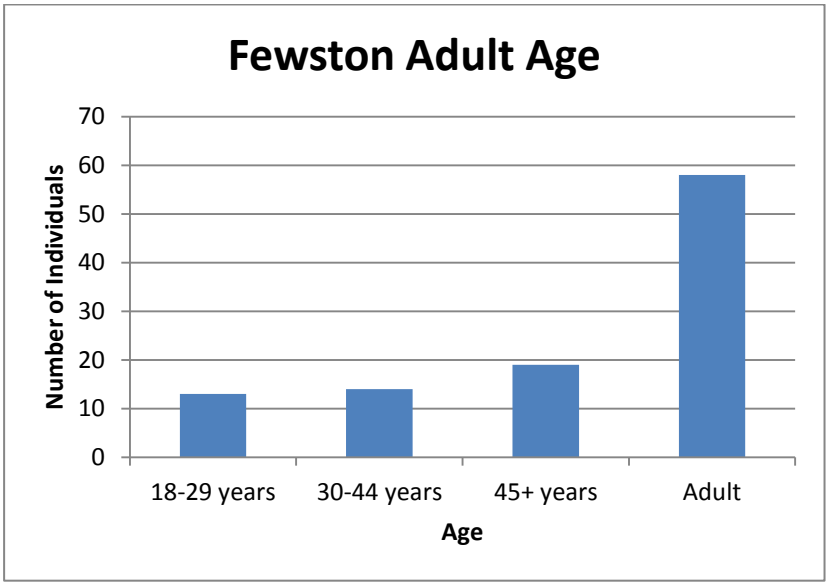

Figure 4.8: Age at death groups of the church of St Michael and St Lawrence, Fewston adults. Adult denotes that no age other than 18+ years old could be determined.

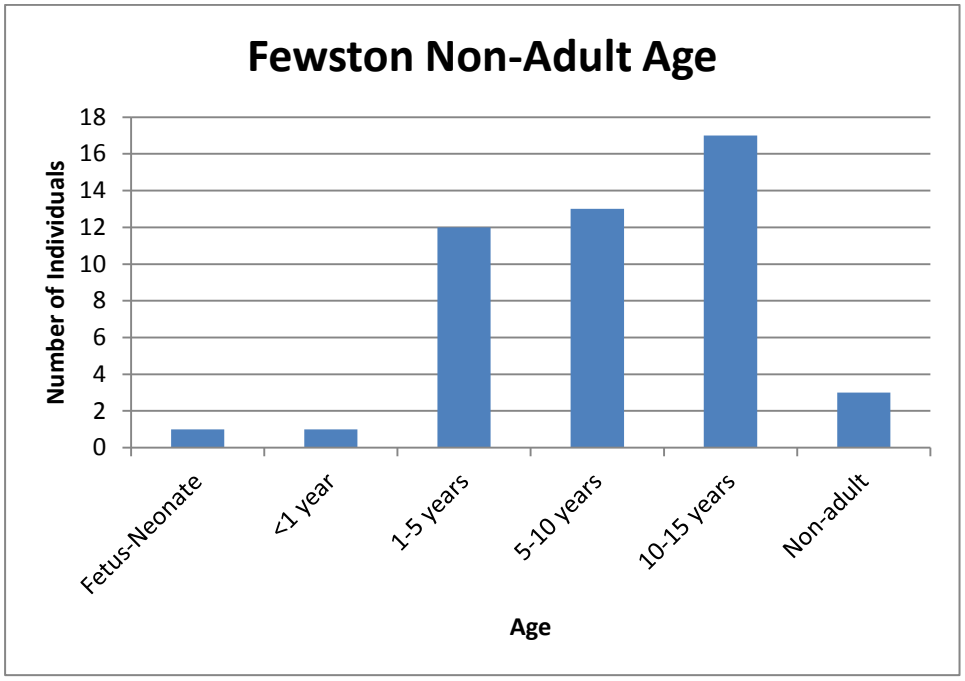

Figure 4.9: Age at death groups of the church of St Michael and St Lawrence, Fewston non-adults. Non-adult denotes no age more specific than <18 years could be determined.

4.2.4 Wharram Percy

In total, 60 individuals were examined from the cemetery of St Martin's church, Wharram Percy dated from 1540 to 1850. Of these individuals, 49 were adults (81.7%) while 11 were non-adults (18.3%). Of the adults, 22 were male, 4 were possibly male (53.1% M/?M), 11 were female, 7 were possibly female (36.7% F/?F), and 5 could not be sexed (10.2% U) (Figure 4.10). The age structure for both adults and non-adults can be found in Figures 4.11 and 4.12.

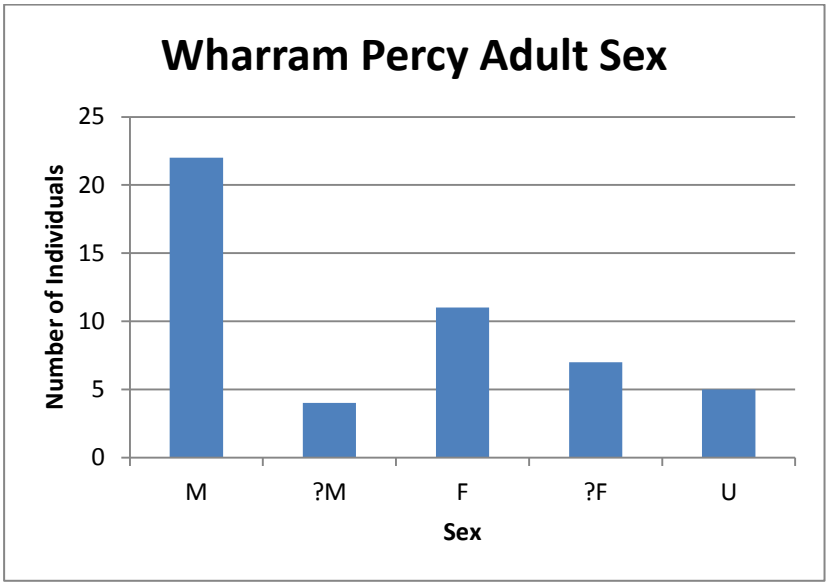

Figure 4.10: Sex breakdown of all adults from St Martin's church, Wharram Percy. M = male; ?M = possible male; F = female; ?F = possible female; U = sex was undeterminable

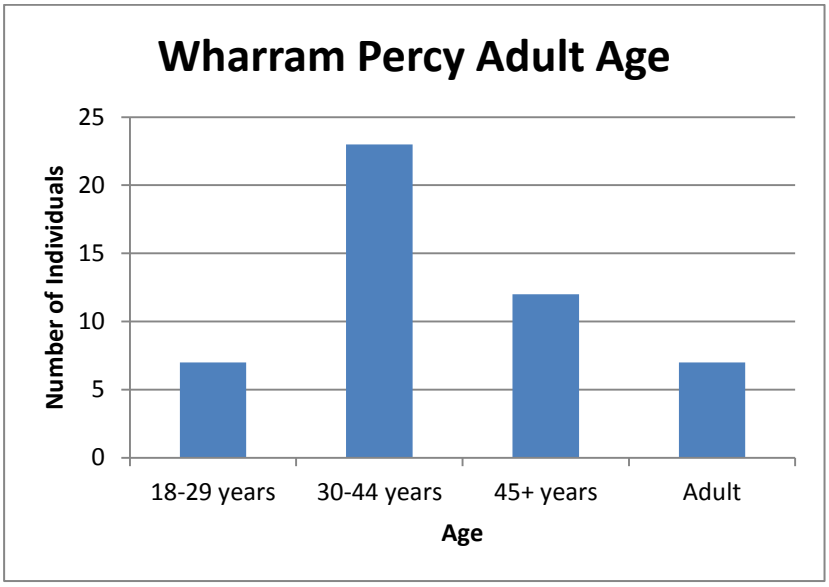

Figure 4.11: Age at death groups of the St Martin's church, Wharram Percy adults. Adult denotes that no age other than 18+ years old could be determined.

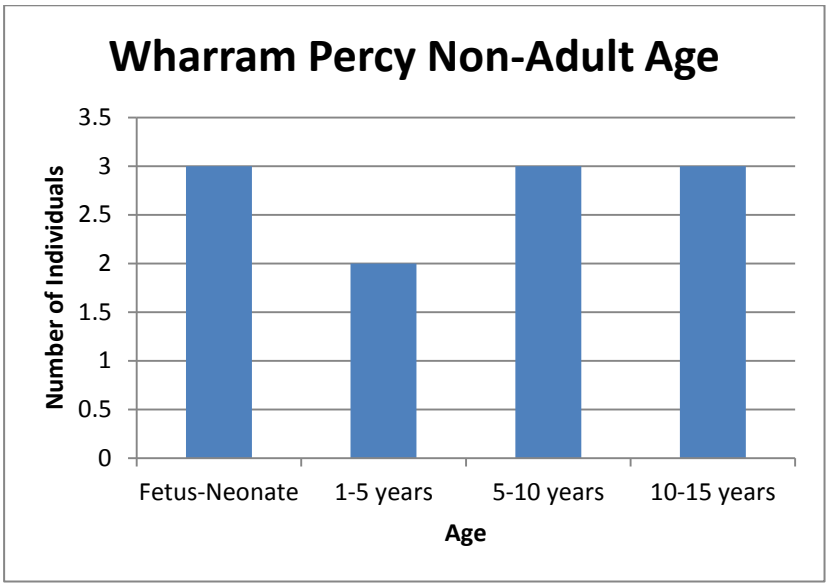

Figure 4.12: Age at death groups of the St Martin's church, Wharram Percy non-adults.

4.3 Congenital Defect Frequency

For all congenital defects combined in all individuals, the prevalence rate for all sites together was 30.15% (Table 4.1, Figure 4.13). Comparing prevalence rates between combined urban and rural sites, there was nearly a statistically significant result (p=0.0846, significant at 90%) with more defects occurring in individuals from urban sites. People buried at Fewston were found to have statistically significantly fewer defects than any other site or the urban sites combined (Table 4. 2). People buried at Wharram Percy had nearly statistically more congenital defects than South Shields (p=0.0876), while North Shields had nearly statistically significantly more congenital defects than the rural sites combined (p=0.0808). The highest number of anomalies present in a single person was five, seen in two individuals, one from Fewston (F366) and one from North Shields (COL10 064). For those individuals with at least one anomaly present, there was no statistical difference found between having one, or having more than one, anomaly. When the sites were compared looking at one or more than one anomaly, the only significantly different result was between North and South Shields. There were statistically more individuals with only one anomaly at North Shields than at South Shields, but this is reversed for individuals with more than one anomaly.

Site		Present	Individuals	CPR TPR TPR
All Sites	ALL	199	660	30.15%
	1	101	199	50.75%
	>1	98	199	49.25%
Fewston	ALL	28	151	18.54%
	1	15	28	53.57%
	>1	13	28	46.43%
Wharram Percy	ALL	26	60	43.33%
	1	11	26	42.31%
	>1	15	26	57.69%
North Shields	ALL	82	245	33.47%
	1	50	82	60.98%
	>1	32	82	39.02%
South Shields	ALL	63	204	30.39%
	1	25	63	39.68%
	>1	38	63	60.32%
Rural	ALL	54	211	25.59%
	1	26	54	48.15%
	>1	28	54	51.85%
Urban	ALL	145	449	32.07%
	1	75	145	51.72%
	>1	70	145	48.28%

Table 4.1: Presence of congenital defects as a whole for all individuals. Present = number of individuals affected in the population; Individuals = number of individuals in the population; ALL = all individuals in the study; 1 - has only one defect present, of the total number of individuals with at least one defect present; >1 - has more than one defect present of the total number of individuals with at least one defect present

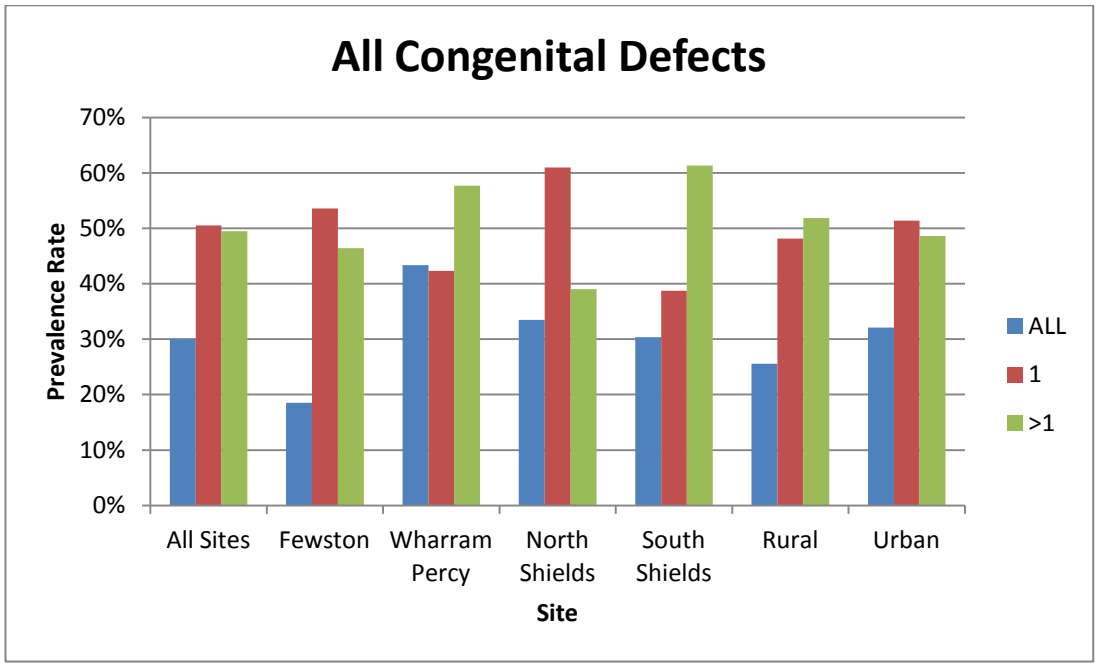

Figure 4.13: Figure showing the crude prevalence rates for the presence of congenital defects for the whole population. ALL = has at least one defect; 1 - has only one defect present, of the total number of individuals with at least one defect present; >1 - has more than one defect present of the total number of individuals with at least one defect present

Site		vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	ALL	0.0004	0.0012	0.0097	-	0.0012
	1	0.4297	0.5119	0.2567	-	1.0000
	>1	0.4297	0.5119	0.2567	-	1.0000
Wharram Percy	ALL	-	0.1757	0.0876	-	0.1089
	1	-	0.1146	0.8172	-	0.4022
	>1	-	0.1146	0.8172	-	0.4022
North Shields	ALL	-	-	0.6125	0.0808	-
	1	-	-	0.0125	0.1603	-
	>1	-	-	0.0125	0.1603	-
South Shields	ALL	-	-	-	0.2752	-
	1	-	-	-	0.4547	-
	>1	-	-	-	0.4547	-
Rural	ALL	-	-	-	-	0.0846
	1	-	-	-	-	0.7501
	>1	-	-	-	-	0.7501

Table 4.2: Statistical analysis (two tailed p values) of the presence of all congenital defects. Yellow indicates a significant result ($p \leq 0.05$) while green indicates a nearly significant result ($p \leq 0.10$).

Some congenital defects are only observable bioarchaeologically after the fusion of epiphyses has taken place (*e.g.* cleft neural arch (see Section 2.4.4) and os acromiale (see Section 2.4.17)). Additionally, hypoplastic or rudimentary defects may be difficult to diagnose in young non-adults due to the small nature of their

bones. As such, it merits looking at congenital defects in only older individuals. Results of looking at congenital defects in only individuals over eighteen years of age at death and for individuals over the age of four at death can be found in Tables 4.3, 4.4, and 4.5. Four years of age was chosen as a cut off point for the second set of results since that is the age the bodies and neural arches of the vertebrae should be fused.

Site	Present	Adults	CPR
All Sites	177	443	39.95%
Fewston	26	104	25.00%
Wharram Percy	25	49	51.02%
North Shields	68	162	41.98%
South Shields	58	128	45.31%
Rural	51	153	33.33%
Urban	126	290	43.45%

Table 4.3: Presence of all congenital defects as a whole for all adult individuals (18+ years old). Present = number of individuals affected in population; Adults = number of individuals in population eighteen or older at the time of death

Site	Present	> 4 Years	CPR
All Sites	195	529	36.86%
Fewston	28	139	20.14%
Wharram Percy	26	55	47.27%
North Shields	79	189	41.80%
South Shields	62	146	42.47%
Rural	54	194	27.84%
Urban	141	335	42.09%

Table 4.4: Presence of all congenital defects as a whole for all individuals over the age of four. Present = number of individuals affected in the population; >4 Years = number of individuals in the population older than four years at the time of death

Site		vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	Adult	0.0019	0.0057	0.0016	-	0.0010
	> 4 Yrs	0.0003	0.0001	0.0001	-	0.0001
Wharram Percy	Adult	-	0.3247	0.5062	-	0.3533
	> 4 Yrs	-	0.5366	0.6327	-	0.5568
North Shields	Adult	-	-	0.6335	0.1309	-
	> 4 Yrs	-	-	0.9116	0.0052	-
South Shields	Adult	-	-	-	0.0491	-
	> 4 Yrs	-	-	-	0.0056	-
Rural	Adult	-	-	-	-	0.0417
	> 4 Yrs	-	-	-	-	0.0011

Table 4.5: Statistical analysis (two tailed p values) of the presence of all congenital defects in adults (18+ years old) and individuals over the age of four. Yellow indicates a significant result ($p \leq 0.05$).

As can be seen, the overall prevalence rates for both age restricted studies are slightly higher than that found for all individuals. For both age restricted groups, individuals at Wharram Percy had the highest rates of congenital defects, while individuals from Fewston had the lowest. Similar to the results for non-age restricted individuals, both adults and those over the age of four years from Fewston showed a statistically significantly lower prevalence rate when compared to each individual site, and to the urban sites combined. For individuals over the age of four, there was a statistically significant higher prevalence of defects at North Shields compared to the combined rural sites (p=0.0052), but this was not seen in the adults only group. At South Shields the adults and the individuals over the age of four years had a significantly higher rate of defects than the combined rural sites (p=0.0491, p=0.0056). The biggest difference between the non-age restricted group and the two age restricted groups comes from the comparison of combined rural versus combined urban sites. In both adults and individuals over the age of four, there is a statistically significant difference (p=0.0417, p=0.0011). In both instances, there were more defects amongst the urban populations.

4.3.1 Craniosynostosis

Seven individuals were recorded as having some sort of asymmetry of the cranium. Two of these individuals, both from Fewston, were diagnosed with craniosynostosis. The first individual, F186 (M, 45+ years old), exhibited partial premature fusion of the right lambdoidal suture (Figure 4.14). The coronal suture ran straight from the sagittal suture to the right side, but ran posteriorly as it moved left from the sagittal suture. The frontal bone projected more anteriorly on the right side while the occipital bone projected more posteriorly on the left side. The facial region was broken post-mortem. The second individual diagnosed with craniosynostosis was F351 another male over 45 years of age. This individual's cranium showed premature fusion of the squamosal and occipito-mastoid sutures on the right side (Figure 2.9). This lead to a slight asymmetry where the left parietal bone projected further than the right.

Figure 4.14: Craniosynostosis due to partial premature closure of the lambdoidal suture on the right side in a male over 45 years old. Areas of note are the diagonally running coronal suture, right side projection of the frontal bone, and left side projection of the occipital. (**A**) Showing entire superior view of cranium. (**B**) Showing a zoomed in image of the anterior portion of the cranium with asymmetry of the frontal bone. (F186, Fewston)

The other individuals with abnormally shaped crania were diagnosed with an asymmetrical cranium as there was no obvious involvement of early suture closure. In total, one individual from Fewston, three from North Shields, and one from South Shields were affected. The individual from Fewston, F165 (M, 45+ years), had the most dramatic changes of the group. The facial region all the way down through the basilar process of the cranium was turned towards the right. The foramen magnum was also skewed to the right. The coronal suture appeared to be more posteriorly located than normal. Moving posteriorly along the cranium, the occipital sulcus and crest leaned to the right and, moving anteriorly, the sagittal sulcus and frontal crest leaned to the left. In addition to cranial asymmetry, mandibular asymmetry was recorded for this individual. The left ramus was noticeably shorter than the right (R=50.3mm, L=46.9mm). The mandibular condyle was smaller on the left (R=17.8mm, L=14.2mm) and its superior surface was porotic, with a matching porotic area on the left mandibular fossa. The ramus and coronoid process angled laterally on the right but were nearly straight on the left. Two of the individuals from North Shields were affected in the occipital region. In COL10 026, a possible male of 35-44 years, the sulcus veered to the right from the lambdoidal suture. Both the internal and external occipital protuberances were

slightly off centre, to the right. In COL10 141, an adult of undetermined sex, the external occipital protuberance was skewed to the right and protruded farther on that side. The internal occipital sulcus curved to the left. The cerebral fossa was much larger on the right side. No other observations could be made as the area below the internal occipital protuberance was missing post-mortem. The third individual from North Shields, COL10 191C, had more widespread changes. In this adult male, the parietal eminence was lower on the right side. There was greater wear on the right mandibular permanent first premolar and first and second molars compared to the molars on the left. Unfortunately, the maxillae were missing post-mortem so it was impossible to see if the same pattern held true on the upper dentition in an effort to attribute this to the cranial asymmetry. The left mandibular condyle and temporo-mandibular articular surface were much larger on the left side. The only affected individual at South Shields was CS06 274, a female over the age of 40. In this instance, the left parietal bone was larger than the right. Both the lambdoidal suture and parietal boss were lower on the left, and the left side of the occipital bone protruded further than the right.

4.3.2 Cleft Lip and Palate

In total, 388 individuals were observable for clefting of the lip, palate, or both (64 at Fewston, 31 at Wharram Percy, 158 at North Shields, and 135 at South Shields). For clefting of the lip only, 381 individuals were observable (58 at Fewston, 31 at Wharram Percy, 157 at North Shields, and 135 at South Shields). For clefting of the palate, 364 individuals were observable (64 at Fewston, 29 at Wharram Percy, 146 at North Shields, and 125 at South Shields). There were no occurrences of cleft lip or cleft palate at any of the study sites.

4.3.3 Elongated Styloid Process

Four individuals from three sites had an elongated styloid process: F138B, COL10 211, CS06 100, and CS06 332. The individual from Fewston was a male over 45 and was only tentatively bilaterally diagnosed. The right process measured 20.8mm and the left measured 25.8mm but both were broken post-mortem. Both

styloid processes were smooth and angled anteriorly. As this appearance is the same as that found in elongated styloid processes, and the true length of the processes is unknown, this individual was given a tentative diagnosis for this defect. North Shields had one individual (COL10 211), an adult possible female (Figure 4.15). The left styloid process measured 34.5mm but the end was broken off post-mortem. The process was angled antero-laterally but was slightly "wavy" in appearance. The right side was broken off and missing post-mortem so laterality could not be determined. Two individuals were found at South Shields with the defect. The first, CO06 100, a male 35-39 years old, was affected bilaterally. The right side measured 33.5mm with the end broken off post-mortem and the left side measured 46.5mm and was intact. The right process was angled anteriorly and the left was angled antero-medially. Both were largely smooth in appearance. The second individual, CS06 332, was a male 30-39 years old. The right styloid process measured 53.3mm and was intact (Figure 2.13). It pointed posteriorly. The left side was missing post-mortem so laterality could not be determined.

Figure 4.15: Elongated styloid process on the left temporal bone of an adult possible female measuring 34.5mm. Anterior is to the right and inferior is at the top of the image. (COL10 211, North Shields)

4.3.4 Cleft Neural Arch

All vertebral types had at least one example of clefting of the posterior neural arch. Examples of cleft neural arch found in the study populations can be found in Figure 4.16. In the cervical vertebrae the first cervical vertebra was clefted five times (F366, COL10 064, COL10 125, COL10 164, CN05) while the seventh was

clefted once in an adult possible male from North Shields (COL10 102) (Table 4.6). Three males, one female, and one possible female were affected by clefting at C1. The fewest number of clefts occurred in the thoracic vertebrae, with only one cleft at T1 in a male 30-34 years old from South Shields (CS06 430) (Table 4.7). The most common location was in the lumbar vertebrae where clefting occurred six times at both L5 and L6 (Table 4.8). Clefting at L5 was seen in five non-adults (COL10 013, COL10 057, COL10 107, COL10 199SC, CS06 984) and one adult female (CS06 344). Clefting at L6 was seen in three males (F366, COL10 265, CS06 371), one possible female (CS06 947), one adult of undetermined sex (CS06 122), and one non-adult (COL10 252). In the sacral segments, S2 was clefted twice, both at North Shields (COL10 148, 15-18 years, and COL10 162, M, 45+ years), and S3 was clefted eight times (Table 4.9). Two males (COL10 083, CN43), two females (CS06 300 and 344), one possible female (CN07), and three non-adults (COL10 148, CS06 096, CS06 228) where affected with clefting at S3. While clefting at S1, S4, and/or S5 is considered normal (the sacral hiatus), these traits were recorded for the purposes of being able to compare this study with other previously published research where this was not considered normal (Table 4.10). None of the comparisons proved to be statistically significant.

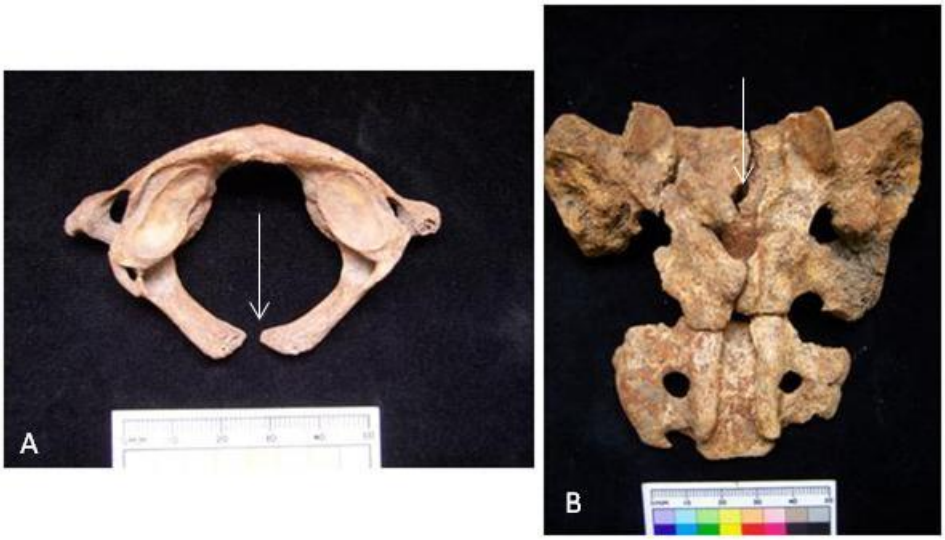

Figure 4.16: (**A**) Cleft neural arch of the first cervical vertebra in a female 30-39 years old. (COL10 164, North Shields) (**B**) Cleft neural arch of the entire sacrum (S1-S5) in a 15-18 year old. (COL10 148, North Shields)

Site		Cleft	Observable	TPR
All Sites	ALL	6	1858	0.32%
	C1	5	278	1.80%
	C7	1	280	0.36%
Fewston	ALL	1	341	0.29%
	C1	1	59	1.69%
	C7	0	42	0%
Wharram Percy	ALL	1	181	0.55%
	C1	1	25	4.00%
	C7	0	26	0%
North Shields	ALL	4	829	0.48%
	C1	3	116	2.59%
	C7	1	126	0.79%
South Shields	ALL	0	507	0%
	C1	0	78	0%
	C7	0	86	0%
Rural	ALL	2	522	0.38%
	C1	2	84	2.38%
	C7	0	68	0%
Urban	ALL	4	1336	0.30%
	C1	3	194	1.55%
	C7	1	212	0.47%

Table 4.6: Presence of cleft cervical vertebrae by location out of the total number of cervical vertebrae observable for the defect.

Site		Cleft	Observable	TPR
All Sites	ALL	1	3491	0.03%
	T1	1	310	0.32%
Fewston	ALL	0	512	0%
	T1	0	45	0%
Wharram Percy	ALL	0	426	0%
	T1	0	34	0%
North Shields	ALL	0	1527	0%
	T1	0	138	0%
South Shields	ALL	1	1026	0.10%
	T1	1	93	1.08%
Rural	ALL	0	938	0%
	T1	0	79	0%
Urban	ALL	1	2553	0.04%
	T1	1	231	0.43%

Table 4.7: Presence and location of cleft thoracic vertebrae out of the total number of thoracic vertebrae observable for the defect.

Site		Cleft	Observable	TPR
All Sites	ALL	12	1542	0.78%
	L5	6	280	2.14%
	L6	6	33	18.18%
Fewston	ALL	1	208	0.48%
	L5	0	31	0%
	L6	1	6	16.67%
Wharram Percy	ALL	0	186	0%
	L5	0	36	0%
	L6	0	4	0%
North Shields	ALL	6	671	0.89%
	L5	4	120	3.33%
	L6	2	7	28.57%
South Shields	ALL	5	477	1.05%
	L5	2	93	2.15%
	L6	3	16	18.75%
Rural	ALL	1	394	0.25%
	L5	0	67	0%
	L6	1	10	10.00%
Urban	ALL	11	1148	0.96%
	L5	6	213	2.82%
	L6	5	23	21.74%

Table 4.8: Presence and location of cleft lumbar vertebrae out of the total number of lumbar vertebrae observable for the defect.

Site		Cleft	Observable	TPR
All Sites	S2	2	195	1.03%
	S3	8	159	5.03%
Fewston	S2	0	26	0%
	S3	0	18	0%
Wharram Percy	S2	0	30	0%
	S3	2	29	6.90%
North Shields	S2	2	73	2.74%
	S3	2	55	3.64%
South Shields	S2	0	66	0%
	S3	4	57	7.02%
Rural	S2	0	56	0%
	S3	2	47	4.26%
Urban	S2	2	139	1.44%
	S3	6	112	5.36%

Table 4.9: Presence of cleft sacral vertebrae, excluding the normal sacral hiatus (clefting at S1, S4, or S5).

Site	Cleft	Observable	TPR
All Sites	45	570	7.89%
Fewston	3	72	4.17%
Wharram Percy	7	94	7.45%
North Shields	21	207	10.14%
South Shields	14	197	7.11%
Rural	10	166	6.02%
Urban	35	404	8.66%

Table 4.10: Presence of clefting at S1, S2, or S3.

Additionally, clefting was considered for the spine as a whole (Tables 4.11 and 4.12). For spinal elements occurring pre-sacrally, there were no statistically significant results. Similarly, for all spinal elements including the sacra (clefting at S1, S4, and/or S5 considered normal) there were no statistically significant outcomes.

Site	Cleft	Observable	TPR	Location
All Sites	20	6891	0.29%	(5x C1, 1x C7, 1x T1, 7x L5, 6x L6)
Fewston	2	1061	0.19%	(1x C1, 1x L6)
Wharram Percy	1	793	0.13%	(1x C1)
North Shields	10	3027	0.33%	(3x C1, 1x C7, 4x L5, 2x L6)
South Shields	7	2010	0.35%	(1x T1, 3x L5, 3x L6)
Rural	3	1854	0.16%	(2x C1, 1x L6)
Urban	17	5037	0.34%	(3x C1, 1x C7, 1x T1, 7x L5, 5x L6)

Table 4.11: Presence of clefting in the pre-sacral spine.

Site	Cleft	Observable	TPR	Location
All Sites	30	7461	0.40%	(5x C1,1x C7, 1x T1, 7x L5, 6x L6, 2x S2, 8x S3)
Fewston	2	1133	0.18%	(1x C1, 1x L6)
Wharram Percy	3	887	0.34%	(1x C1, 2x S3)
North Shields	14	3234	0.43%	(3x C1, 1x C7, 4x L5, 2x L6, 2x S2, 2x S3)
South Shields	11	2207	0.50%	(1x T1, 3x L5, 3x L6, 4x S3)
Rural	5	2020	0.25%	(2x C1, 1x L6, 2x S3)
Urban	25	5441	0.46%	(3x C1, 1x C7, 1x T1, 7x L5, 5x L6, 2x S2, 6x S3)

Table 4.12: Presence of clefting occurring anywhere in the spine. Clefting at S1, S4, or S5 is not considered abnormal so is not included in the counts for clefting, but the presence of these elements is included in the observable total.

4.3.5 Hypoplastic and Aplastic Lamina

Ten individuals were found to have a hypoplastic lamina on at least one vertebra. Eight individuals had hypoplastic laminae in the cervical vertebrae, two in the lumbar vertebrae, and one in the sacrum (one individual had this defect in two locations of the spine, see below). In F345 (14-18 years) and COL10 005 (F, Adult), the hypoplastic lamina occurred on the left side of C1 (Figure 4.17A). Other single segment occurrences in the cervical vertebrae were hypoplasia of the right lamina in C4 (COL10 027, ?F, Adult), bilateral hypoplasia in C5 (COL10 113, ?F 30-34 years), and left hypoplasia in C6 (COL10 102, ?M, Adult). In three individuals, both the C2 and C3 were affected. In individual COL10 008 (?M, 40+ years), the right lamina was hypoplastic in C2 while the left lamina was hypoplastic in C3 (Figure 4.17B). The right inferior apophyseal facet was displaced anteriorly and malformed. The complimentary facet in C3 was similarly displaced and flattened. The dens leaned to the right, but in anatomical position was straight due to the C2 "sitting" at an angle on C3. There was a partial cleft in the left lamina of C3. The right laminae were hypoplastic in both C2 and C3 for COL10 064 (?F, 30-39 years). In individual CS06 678 (about 12 years), the right lamina was hypoplastic in C2 and the left was hypoplastic in C3. There were two occurrences of hypoplastic laminae in the lumbar region. A male over 45 years of age showed hypoplasia on the left side in the sacralized sixth lumbar vertebra (F138B). The other hypoplastic lumbar lamina was on the right side of the L5 of COL10 017 (?F, Adult). This individual also accounted for the only occurrence of hypoplastic lamina in the sacrum. The right side was hypoplastic at S1.

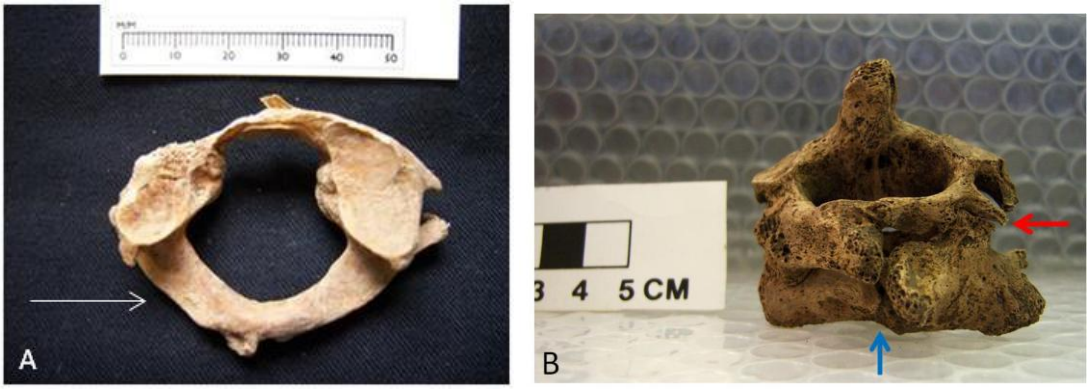

Figure 4.17: (**A**) Left hypoplasia of the lamina of the first cervical vertebra in a 14-18 year old non-adult. (F345, Fewston) (**B**) Hypoplasia of the lamina on the right in C2 and on the left in C3 in a possible male over 40 years old. The red arrow shows the displaced apophyseal facets and the blue arrow shows the partial cleft in C3. (COL10 008, North Shields)

Only two occurrences of aplasia of the lamina were present in the study populations. The left lamina of the first sacral segment was aplastic in F300, a 45+ years old female. The right side was normal, terminating at the midline. Aplasia of the left lamina of course produced a cleft (see Sections 2.4.5 and 3.3.5) so this individual was included in all counts of clefting at the first sacral segment in Section 4.3.4. In the second, the right lamina was aplastic on the seventh cervical vertebra in individual COL10 102, an adult possible male. This defect corresponds with hypoplasia of the left lamina and aplasia of the right inferior apophyseal facet in the sixth cervical vertebra. Again, this defect resulted in a cleft neural arch and this individual was included in counts of clefting in the cervical vertebrae.

4.3.6 Spondylolysis

Spondylolysis was only found in the lumbar vertebrae and was seen in individuals from all sites except for Wharram Percy (Figure 4.18). The number of occurrences and true prevalence rates are displayed in Table 4.13. The fifth lumbar vertebra was the most commonly affected segment with twelve instances, but the second and fourth lumbar vertebrae each displayed the trait once. The affected second lumbar vertebra was from South Shields (CS06 984, 3-4 years) while the affected fourth lumbar vertebra was from North Shields (COL10 093, M, 45+ years). The individuals affected with spondylolysis at L5 were eight males (COL10 093, COL10 115, CS06 125, CS06 215, CS06 371, CS06 390, CS06 492, F366), one possible

male (CS06 965), one female (CS06 532), one possible female (CS06 623), and one non-adult (COL10 107). When considering spondylolysis in the lumbar vertebrae, none of the comparisons of sites proved to be statistically significant (Table 4.14). However, when South Shields was compared to North Shields and to the combined rural populations, the results were nearly statistically significant (p=0.0890 and p=0.0517 respectively), with more spondylolysis occurring at South Shields for both instances.

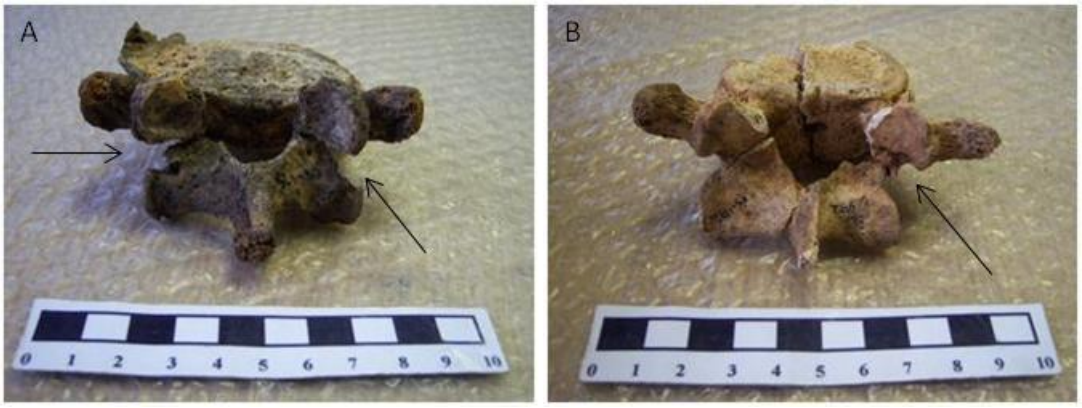

Figure 4.18: (**A**) Bilateral spondylolysis of the fifth lumbar vertebra of male over 40 years old. The separate neural arch is placed in anatomical position. (CS06 125, South Shields) (**B**) Right unilateral spondylolysis of the fifth lumbar vertebra of a male 30-34 years old. The left pars interarticularis was broken post-mortem. (CO06 492, South Shields)

Site		Present	Observable	TPR	Location
All Sites	ALL	14	1356	1.03%	(L2, L4, 12xL5)
	L5	12	266	4.51%	-
Fewston	ALL	1	156	0.64%	(L5)
	L5	1	25	4.00%	-
Wharram Percy	ALL	0	168	0%	-
	L5	0	35	0%	-
North Shields	ALL	4	581	0.69%	(L4, 3xL5)
	L5	3	111	2.70%	-
South Shields	ALL	9	451	2.00%	(L2, 8xL5)
	L5	8	95	8.42%	-
Rural	ALL	1	324	0.31%	(L5)
	L5	1	60	1.67%	-
Urban	ALL	13	1032	1.26%	(L2, L4, 11xL5)
	L5	11	206	5.34%	-

Table 4.13: Presence of spondylolysis in the lumbar vertebrae.

Site		vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	ALL	0.4815	1.0000	0.4656	-	1.0000
	L5	0.4167	0.5607	0.6830	-	1.0000
Wharram Percy	ALL	-	0.5800	0.1227	-	0.2349
	L5	-	1.0000	0.1074	-	0.3743
North Shields	ALL	-	-	0.0890	0.6599	-
	L5	-	-	0.1169	1.0000	-
South Shields	ALL	-	-	-	0.0517	-
	L5	-	-	-	0.1547	-
Rural	ALL	-	-	-	-	0.2088
	L5	-	-	-	-	0.3091

Table 4.14: Statistical analysis (two tailed p values) for the presence of spondylolysis in the lumbar vertebrae. Green indicates a nearly significant result (p≤0.10).

4.3.7 Block Vertebrae/Klippel-Feil Syndrome

Block vertebrae, or congenital fusion outside of the cervical region of the spine, occurred in four individuals, three times in the thoracic vertebrae and once in the lumbar vertebrae (Figure 4.19). Individual CS06 678 (~12years) displayed fusion of T2 and T3 while SA23 (F, 45+ years) displayed fusion of T4 and T5. A neonate (COL10 235) had congenital fusion of two unidentified thoracic vertebrae. Only the right side of the neural arch of the affected vertebrae was recovered during excavation. The sole evidence of congenital fusion to occur in the lumbar region was seen in CS06 390 (M, 40-44 years) between L3 and L4.

Figure 4.19: Block vertebra due to congenital fusion of T2 and T3 in a non-adult about 12 years old. Notice the unilateral (right) fusion of the transverse processes and fusion of the neural arches. (CS06 678, South Shields)

Klippel-Feil syndrome, congenital fusion in the cervical vertebrae, occurred in four individuals, all in different locations of the cervical region (Figure 4.20).

Individual COL10 145 (?M, 30-34 years) showed fusion of C2 and C3, F241 (M, Adult) showed fusion of C3 and C4, COL10 049 (M, 40-44 years) showed fusion at C5 and C6, and COL10 128 (M, 30-34 years) showed fusion at C6 and C7 (Figure 2.18).

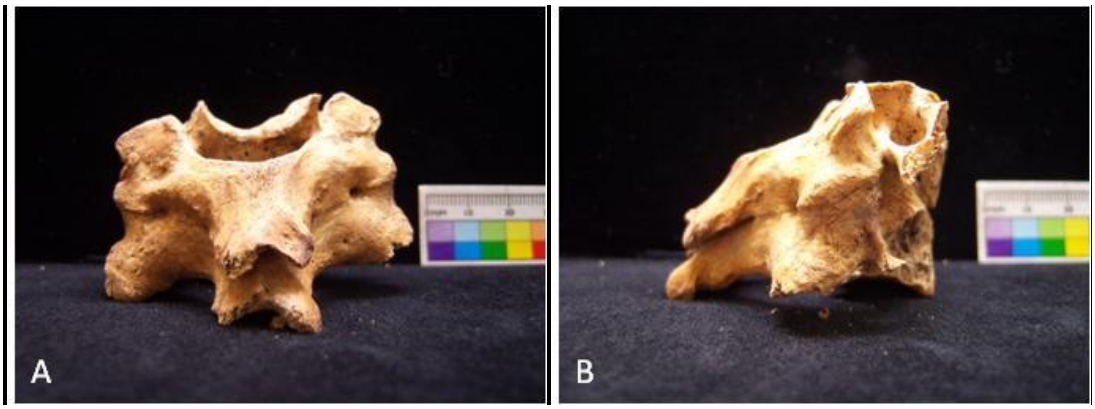

Figure 4.20: Klippel-Feil syndrome causing congenital fusion of C3 and C4 in an adult male. (**A**) Posterior view. (**B**) Right lateral view (F241, Fewston)

4.3.8 Transitional Vertebra

A. Occipitocervical Border

There were no occurrences of an occipital vertebra in the 414 individuals observable (border shift in the cranial direction). In the caudal direction, there was one occurrence of occipitalization (incomplete) of the first cervical vertebra in the 456 individuals observable (TPR=0.22%) (Figure 4.21). This was seen in a male over 45 years old (COL10 125) from North Shields (all individuals 1/456, TPR=0.22%; NS 1/193, TPR=0.52%). No comparisons between sites were statistically significant for the presence of occipitalization. A precondylar facet, a mild expression of caudal shifting, was seen in one individual, skeleton F122 (all individuals 1/456, TPR=0.22%; Fe 1/93, TPR=1.08%) (Figure 2.19B). In this female over 45 years old, the first cervical vertebra was missing post-mortem so it was not possible to observe the full nature of the changes due to this shift. When this defect is combined with occipitalization, the true prevalence rate for caudal shifts was 0.44% (2/456). No comparisons between sites for the presence of a caudal shift were statistically significant.

Figure 4.21: Incomplete occipitalization of the first cervical vertebra in a male over 45 years old. The first cervical vertebra also displays a cleft neural arch. (COL10 125, North Shields)

B. Cervicothoracic Border

Cervical ribs were found in association with the seventh cervical vertebra in eight individuals. The population buried at North Shields had the most occurrences (four). Skeletons COL10 087 (M, 17-24 years) and 125 (M, 45+ years) had bilateral involvement, while skeletons COL10 170 (?M, 20-24 years) and 216 (M, 35-39 years) were affected on the right side, but the left sides were unobservable for the condition. Two cervical ribs were found in skeletons CS06 264 (?M, 45+ years) and 987 (M, 30-34 years) at South Shields. Both occurred on the right side although the left sides were broken and missing postmortem and therefore unobservable. One occurrence each was found at Fewston and Wharram Percy. The Fewston individual (F062, 10-11 years) had a cervical rib on the right side, but the left side was damaged postmortem. At Wharram Percy (V42, M, 40-44 years), the rib again occurred on the right side but in this instance the left side was normal (Figure 4.22).

Figure 4.22: Cervical rib on the right side of the seventh vertebra in a male 40-44 years old. (V42, Wharram Percy)

C. Thoracolumbar Border

Shifts at the thoracolumbar border were the most commonly occurring congenital defect. The crude prevalence rate was 13.48% for all shifts at this location, with 11.21% for cranial shifts (lumbarization of T12), and 2.27% for caudal shifts (lumbar ribs, although not all exhibited costal facets or ribs) (Figure 4.23, Table 4.15). Two different true prevalence rates were examined. The first was that for individuals with T11, T12, T13, or L1 present (Table 4.16). The prevalence rates were 27.47% for all shifts, 22.84% for cranial shifts, and 4.63% for caudal shifts. The second was that for all individuals with a completely preserved spine which showed prevalence rates of 33.33% for all shifts, 28.93% for cranial shifts, and 4.40% for caudal shifts (Table 4.17).

Figure 4.23: (**A**) Costal facet for a lumbar rib on the right side of the first lumbar vertebra in a male over 45 years old. (CS06 135, South Shields) (**B**) Partial lumbarization of the twelfth thoracic vertebra in a possible male 18-23 years old. The left superior apophyseal facet is normal but the right has rotated to face medially (arrow). (WCO010, Wharram Percy)

Site		Present	Individuals	Total Shifts	CPR 1	CPR 2
All Sites	ALL	89	660	-	13.48%	-
	CRANIAL	74	660	89	11.21%	83.15%
	CAUDAL	15	660	89	2.27%	16.85%
Fewston	ALL	9	151	-	5.96%	-
	CRANIAL	4	151	9	2.65%	44.44%
	CAUDAL	5	151	9	3.31%	55.56%
Wharram Percy	ALL	12	60	-	20.00%	-
	CRANIAL	11	60	12	18.33%	91.67%
	CAUDAL	1	60	12	1.67%	8.33%
North Shields	ALL	39	245	-	15.92%	-
	CRANIAL	34	245	39	13.88%	87.18%
	CAUDAL	5	245	39	2.04%	12.82%
South Shields	ALL	29	204	-	14.22%	-
	CRANIAL	25	204	29	12.25%	86.21%
	CAUDAL	4	204	29	01.96%	13.79%
Rural	ALL	21	211	-	9.95%	-
	CRANIAL	15	211	21	7.11%	71.43%
	CAUDAL	6	211	21	2.84%	28.57%
Urban	ALL	68	449	-	15.14%	-
	CRANIAL	59	449	68	13.14%	86.76%
	CAUDAL	9	449	68	2.00%	13.24%

Table 4.15: Presence of shifting at the thoracolumbar border. CPR 1 - prevalence of the trait in all individuals observed; CPR 2 - prevalence of the direction of shifting in all individuals with a shift at this location

Site		Present	Individuals	TPR
All Sites	ALL	89	324	27.47%
	CRANIAL	74	324	22.84%
	CAUDAL	15	324	4.63%
Fewston	ALL	9	44	20.45%
	CRANIAL	4	44	9.09%
	CAUDAL	5	44	11.36%
Wharram Percy	ALL	12	39	30.77%
	CRANIAL	11	39	28.21%
	CAUDAL	1	39	2.56%
North Shields	ALL	39	144	27.08%
	CRANIAL	34	144	23.61%
	CAUDAL	5	144	3.47%
South Shields	ALL	29	97	29.90%
	CRANIAL	25	97	25.77%
	CAUDAL	4	97	4.12%
Rural	ALL	21	83	25.30%
	CRANIAL	15	83	18.07%
	CAUDAL	6	83	7.23%
Urban	ALL	68	241	28.22%
	CRANIAL	59	241	24.48%
	CAUDAL	9	241	3.73%

Table 4.16: Presence of shifting at the thoracolumbar border for individuals with T11, T12, T13, or L1 present.

Site		Present	Individuals	TPR
All Sites	ALL	53	159	33.33%
	CRANIAL	46	159	28.93%
	CAUDAL	7	159	4.40%
Fewston	ALL	1	18	5.56%
	CRANIAL	1	18	5.56%
	CAUDAL	0	18	0.00%
Wharram Percy	ALL	5	15	33.33%
	CRANIAL	4	15	26.67%
	CAUDAL	1	15	6.67%
North Shields	ALL	29	79	36.71%
	CRANIAL	25	79	31.65%
	CAUDAL	4	79	5.06%
South Shields	ALL	18	47	38.30%
	CRANIAL	16	47	34.04%
	CAUDAL	2	47	4.26%
Rural	ALL	6	33	18.18%
	CRANIAL	5	33	15.15%
	CAUDAL	1	33	3.03%
Urban	ALL	47	126	37.30%
	CRANIAL	41	126	32.54%
	CAUDAL	6	126	4.76%

Table 4.17: Presence of shifting at the thoracolumbar border for individuals with complete spines.

D. Lumbosacral Border

A caudal shift at the lumbosacral border, lumbarization, only occurred in five individuals resulting in a 0.76% crude prevalence rate or a 1.47% true prevalence rate (calculated for individuals with S1 and/or S2 present) (Figure 4.24, Table 4.18). Two males (F351, COL10 176), one female (CS06 479), and two possible females (F098, F348) were affected. When looking at the crude prevalence rates, no differences between sites were found to be statistically significant. For true prevalence rates, Fewston had statistically more affected individuals than North Shields (p=0.0432) and urban sites (p=0.0262) combined, and had nearly more statistically significant occurrences than in individuals from South Shields (p=0753). No other comparisons proved to be statistically significant.

Figure 4.24: Lumbarization of the first sacral segment leaving behind only four sacral segments in a possible female 17-25 years old. (F348, Fewston)

Site		Present	Individuals	Prevalence Rates	Description
All Sites	C	5	660	0.76%	
	T	5	339	1.47%	
Fewston	C	3	151	1.99%	(S1 complete, 2x S1 incomplete)
	T	3	45	6.67%	
Wharram Percy	C	0	60	0%	
	T	0	42	0%	
North Shields	C	1	245	0.41%	(S1 complete)
	T	1	143	0.70%	
South Shields	C	1	204	0.49%	(S1 incomplete)
	T	1	109	0.92%	
Rural	C	3	211	1.42%	
	T	3	87	3.45%	
Urban	C	2	449	0.45%	
	T	2	252	0.79%	

Table 4.18: Presence of lumbarization at the lumbosacral border. C - crude prevalence; T - true prevalence

Forty eight individuals were found to exhibit sacralization, a cranial shift of the lumbosacral border, resulting in a 7.27% crude prevalence rate (Figure 4.25, Table 4.19). Affected were twenty males, five possible males, nine females, ten possible females, three adults of undetermined sex, and one non-adult. Individuals buried at South Shields had statistically significantly more occurrences of sacralization than people either at Fewston (p=0.0144) or North Shields (p=0.0010). Individuals buried at Wharram Percy also had statistically more occurrences of this feature than those buried at North Shields (p=0.0213). The data from Wharram

Percy were nearly more statistically significant than from Fewston (p=0.0734) while those from South Shields was nearly more statistically significant than the combined rural sites (p=0.0635). Further break down by vertebral element affected can be found below (*NB*: One instance of partial sacralization on the right side in an individual from Wharram Percy was diagnosed due to the changes observed on the first sacral element. All lumbar vertebrae were missing post-mortem. In this situation, it is impossible to know for certain whether the affected element was in fact an L5 and not an L6, and therefore it is not mentioned below).

Figure 4.25: Complete sacralization of the sixth lumbar vertebra in a male 35-39 years old. (CS06 323, South Shields)

Of the 339 L5 vertebrae present, fifteen were sacralized (4.42%). The most common form was complete sacralization, occurring in twelve individuals. There was one instance of incomplete sacralization on the right side and one instance of partial sacralization for each side. When comparing the prevalence for the occurrence of this trait at L5, it was proven statistically significant that non-sacralization occurred more commonly (p=0.0005). Individuals buried at North Shields had statistically fewer occurrences of this condition than at South Shields (p=0.0106) and nearly statistically fewer occurrences than those buried at Wharram Percy (p=0.0630) (Table 4.20).

Site		Present	Individuals	CPR TPR TPR
All Sites	ALL	48	660	7.27%
	L5	15	339	4.42%
	L6	32	38	84.21%
Fewston	ALL	7	151	4.64%
	L5	1	43	2.33%
	L6	6	6	100.00%
Wharram Percy	ALL	7	60	11.67%
	L5	3	39	7.69%
	L6	3	6	50.00%
North Shields	ALL	9	245	3.67%
	L5	2	147	1.36%
	L6	7	10	70.00%
South Shields	ALL	25	204	12.25%
	L5	9	110	8.18%
	L6	16	16	100.00%
Rural	ALL	14	211	6.64%
	L5	4	82	4.88%
	L6	9	12	75.00%
Urban	ALL	34	449	7.57%
	L5	11	257	4.28%
	L6	23	26	88.46%

Table 4.19: Presence of sacralization comparing location. One instance from Wharram Percy has not been assigned a location due to no lumbar vertebrae being preserved. The diagnosis was made based on changes to the sacrum.

Site		vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	ALL	0.0734	0.6123	0.0144	-	0.2651
	L5	0.3423	0.5390	0.2840	-	1.0000
Wharram Percy	ALL	-	0.0213	1.0000	-	0.3082
	L5	-	0.06300	1.0000	-	0.4076
North Shields	ALL	-	-	0.0010	0.1977	-
	L5	-	-	0.0106	0.1908	-
South Shields	ALL	-	-	-	0.0635	-
	L5	-	-	-	0.4036	-
Rural	ALL	-	-	-	-	0.7492
	L5	-	-	-	-	0.7640

Table 4.20: Statistical analysis (two tailed p values) for the presence of sacralization. Yellow indicates a significant result ($p \leq 0.05$) while green indicates a nearly significant result ($p \leq 0.10$).

Of the 38 sixth lumbar vertebrae present, thirty six were sacralized (84.21%). This was found to be very statistically significant (p=0.0001). Of these occurrences, 23 were completely sacralized, four were incomplete (once on the right side and three times on the left side), and two were partial (once on each side). Two vertebrae were damaged postmortem so only their left sides were observable. The remaining occurrence was from North Shields, occurring in a 14 to 16 year old where the vertebrae were still in the process of fusing. At that stage, it is difficult to tell if the sacralization would have been complete or incomplete once developed. When looking at the trait by site, all individuals with an L6 were affected with sacralization at both Fewston (six individuals) and South Shields (16 individuals). Seventy percent were affected at North Shields (7/10) and fifty percent were affected at Wharram Percy (3/6).

4.3.9 Numerical Variation in Vertebrae

Additional vertebral segments were found in all areas of the spine except the cervical region. Three individuals possessed a thirteenth thoracic segment, 38 individuals had a sixth lumbar vertebra, and six individuals had a sixth sacral segment (Table 4.21). Thirteen thoracic vertebrae were seen in two males (F119, F459) and one female (CS06 107). For the presence of a sixth lumbar vertebra, sixteen males, four possible males, seven females, six possible females, two adults of undetermined sex, and three non-adults were affected. Three males (COL10 038, COL10 168, CS06 559), two females (CN13, CN41), and one possible female (COL10 094) had a sixth sacral segment present. For all areas of the spine, to determine true prevalence rates, the number of supernumerary vertebrae present was compared to those individuals with that particular segment of the spine complete. For the presence of T13, the data from Fewston proved to be significantly different to that from both North Shields (p=0.0306) and the combined urban populations (p=0.0351) (Table 4.22, Figure 4.26). In both instances, T13 occurred more often at Fewston. Additionally, the data from North Shields were nearly statistically significant compared to the combined rural populations (p=0.0957), with the rural populations having a higher occurrence rate of T13. For the presence of L6, a

comparison of data from North Shields and South Shields proved to be the only statistically significant result (p=0.0187), but the data from North Shields compared to Wharram Percy (p=0.0905) and to the combined rural populations (p=0.0552) was not quite statistically significant (Table 4.23). In all these instances, individuals buried at North Shields had fewer occurrences of L6 compared to the other populations. There were no statistically significant results for the presence of S6.

Site		Present	Individuals	TPR
All Sites	T13	3	226	1.33%
	L6	38	304	12.50%
	S6	6	130	4.62%
Fewston	T13	2	23	8.70%
	L6	6	39	15.38%
	S6	0	11	0%
Wharram Percy	T13	0	25	0%
	L6	6	34	17.65%
	S6	2	28	7.14%
North Shields	T13	0	106	0%
	L6	10	140	7.14%
	S6	3	47	6.38%
South Shields	T13	1	72	1.39%
	L6	16	91	17.58%
	S6	1	44	2.27%
Rural	T13	2	48	4.17%
	L6	12	73	16.44%
	S6	2	39	5.13%
Urban	T13	1	178	0.56%
	L6	26	231	11.26%
	S6	4	91	4.40%

Table 4.21: Presence of supernumerary vertebrae.

Site	vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	0.2243	0.0306	0.1444	-	0.0351
Wharram Percy	-	1.0000	1.0000	-	1.0000
North Shields	-	-	0.4045	0.0957	-
South Shields	-	-	-	0.5631	-
Rural	-	-	-	-	0.1149

Table 4.22: Statistical analysis (two tailed p values) for the presence of a thirteenth thoracic vertebra. Yellow indicates a significant result (p≤0.05) while green indicates a nearly significant result (p≤0.10).

Figure 4.26: Complete spine (although missing C2 post-mortem) showing 13 thoracic vertebrae present in a female over 45 years old. (CS06 107, South Shields)

Site	vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	1.0000	0.1203	1.0000	-	0.4294
Wharram Percy	-	0.0905	1.0000	-	0.2686
North Shields	-	-	0.0187	0.0552	-
South Shields	-	-	-	1.0000	-
Rural	-	-	-	-	0.3090

Table 4.23: Statistical analysis (two tailed p values) for the presence of a sixth lumbar vertebra. Yellow indicates a significant result ($p \leq 0.05$) while green indicates a nearly significant result ($p \leq 0.10$).

Three individuals had a congenitally absent vertebral segment, leading to a total number of only 28 segments for the whole spine. One individual from Wharram Percy, a 12-16 year non-adult (SA45) had only 6 cervical vertebrae present. The other individuals, CS06 974 (M, 25-34 years) and CS06 1010 (M, 30-34 years), both came from South Shields and were both lacking a sacral segment (Figure 4.27).

Figure 4.27: Complete sacrum containing only four segments from a male 30-34 years old. (CS06 1010, South Shields)

4.3.10 Scoliosis

Four individuals had definite or possible idiopathic scoliosis, leading to a prevalence rate of 0.81% (4/493), calculated on the basis of the total number of individuals with at least one thoracic vertebra present. Two individuals were affected at North Shields while one was found at both Fewston and South Shields. Skeleton COL10 078 from North Shields had the most definitive evidence (Figure 4.28). Changes in the spine can be seen from T1 through L1. However, the lower lumbar vertebrae are too damaged post mortem to observe any changes due to scoliosis. The curvature of the spine starts with a curve to the left in the upper thoracic vertebrae, then changes to the right in the middle thoracic vertebrae, followed by a change back to the left in the lower thoracic vertebrae. Changes to the vertebral elements include abnormally thin pedicles on one side, “shifting” of the upper and lower vertebral bodies in different directions, compression of one side of the bodies, and asymmetrical articular facets. Additionally, degenerative joint changes are seen in the middle to lower thoracic vertebrae. Other changes due to scoliosis can be seen in the ribs and clavicles. The ribs on the right side have a much tighter curve compared to those on the left, and several right ribs have accessory articular facets where they articulate with adjacent ribs, due to the impact of unusual compression and orientation caused by the curvature of the

spine. The right clavicle has a compressed appearance when compared to the left side. While similar in length to the left clavicle, the right clavicle has much tighter curves. Radiography of the right clavicle revealed no sign of an ante-mortem fracture.

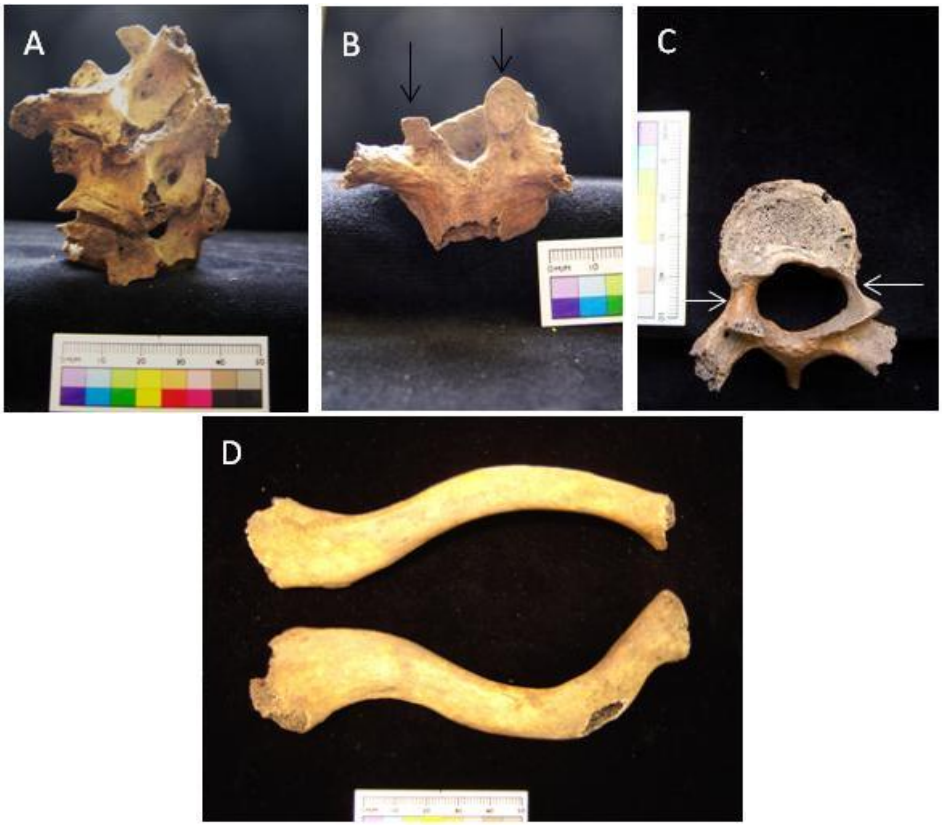

Figure 4.28: Scoliosis as seen in a possible male over 45 years old. (**A**) Showing a curvature to the left in T9, T10, and T11. (**B**) Showing the size discrepancies in superior apophyseal facets and the slanting body of T7. (**C**) Showing the thickness discrepancies in the pedicles and the skewing of the upper body to the left of T3. (**D**) Showing the asymmetry of the clavicles. (COL10 078, North Shields)

The other evidence for scoliosis from North Shields is seen in skeleton COL10 164 (Figure 4.29). Scoliotic changes are seen from C5 to L3. The upper spine curves to the right, then changes to the left at T6/T7, until about T11, where curvature returns to normal. Signs of scoliosis on the vertebral elements include abnormal thinning of the pedicles unilaterally, asymmetrical articular facets, abnormal shape of vertebral foramina (asymmetrical laminae), asymmetrical flattening of the bodies, and asymmetrical neural arches. Degenerative joint changes were also seen in most of the spine at the articular facets. The ribs are too fragmentary to observe for any changes due to scoliosis.

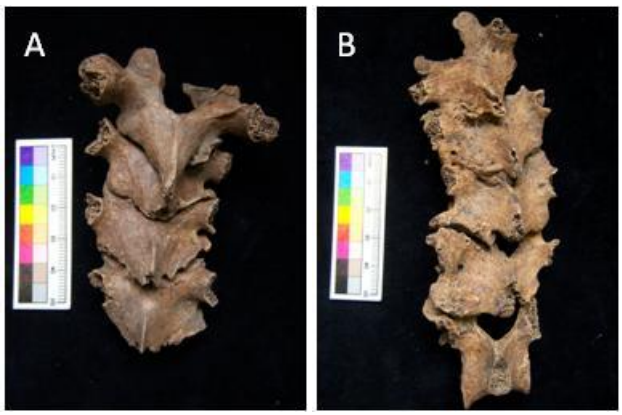

Figure 4.29: Scoliosis in a female 30-39 years old. (**A**) Curvature to the right in T2-T5. (**B**) Curvature to the left in T8-T12. (COL10 164, North Shields)

The spine of skeleton CS06 00315 from South Shields exhibits some changes consistent with idiopathic scoliosis from T3 to T8 (Figure 2.24). The spine curves to the right before correcting itself. Scoliotic changes to the vertebral elements are body compression on one side and thinning of one pedicle relative to the opposite side. Degenerative joint changes are seen on most articular facets of the thoracic spine. The neural arches are fused from T3 to T5 and T6 to T7. Additionally the bodies of T6 and T7 are fused on the left side. Due to postmortem damage and fragmentation, the diagnosis in this individual is not definitive and alternative diagnoses, such as trauma and osteoarthritis or other degenerative joint disease, have to be considered as the causes for these changes. The final and very tentative occurrence of scoliosis comes from Fewston (F289). Two unidentified thoracic bodies show possible signs of scoliosis. The first has a thinner pedicle on the right when compared to the left, and the lower half of the body appears to lean to the right. The second has a thinner pedicle on the left side and the lower half of the body appears to lean to the left. Unfortunately, the remaining four thoracic and five lumbar vertebrae are too poorly preserved and fragmentary to see any other changes that could make this diagnosis more definite.

4.3.11 Rudimentary and Aplastic Transverse Process

At Wharram Percy, one male 40-44 years old (V42) was found to have bilateral rudimentary transverse processes on his first thoracic vertebra (Figure 4.30). The costal facets on the body and the first ribs were normal in appearance.

One occurrence of an aplastic transverse process was found in an individual from South Shields. Skeleton CS06 430 (M, 30-34 years) was lacking the right transverse process on the right side of L1.

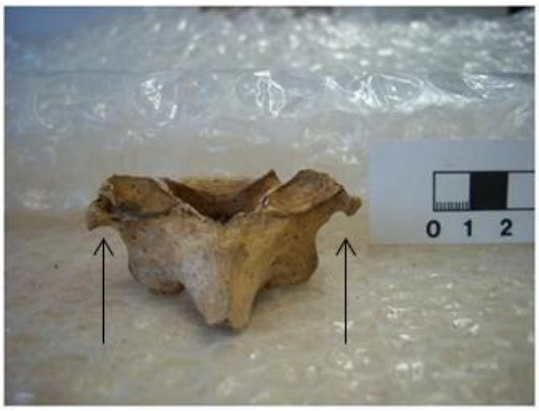

Figure 4.30: Bilateral rudimentary transverse processes on the first thoracic vertebra of a male 40-44 years old. (V42, Wharram Percy)

4.3.12 Rudimentary and Aplastic Apophyseal Facet

Rudimentary apophyseal facets were found in three individuals, two from North Shields and one from South Shields. Skeleton COL10 064 (?F, 30-39 years) showed rudimentary facets on the left side of multiple cervical vertebrae, starting with the inferior apophyseal facet on C2 and ending with the superior apophyseal facet on C6. The other two occurrences appeared at the lumbosacral border. Skeleton COL10 090 (?F, Adult) had rudimentary facets on the right side at the inferior facet of L6 and the superior facet of S1. Skeleton CS06 956 (F, 35-39 years) showed rudimentary facets on the left side of the inferior apophyseal facet of L5 and the superior apophyseal facet of S1.

Only one possible occurrence of aplasia of an apophyseal facet was found in the study populations. The left inferior apophyseal facet was absent from the fifth lumbar vertebra of skeleton CS06 203 (F, 30-39 years) (Figure 4.31). The apophyseal facets of S1 were normal. The area of the lamina where the facet should have been had a porous appearance similar to that seen at the abnormal joint surfaces of os acromiale or spondylolysis, which may imply a failure of fusion (with the separate facet not recovered during excavation) rather than a failure to develop.

Figure 4.31: Aplastic left inferior apophyseal facet of the fifth lumbar vertebra in a female 30-39 years old. (CS06 203, South Shields)

4.3.13 Facet Tropism

Facet tropism was recorded in five individuals, one from Fewston (F186), three from North Shields (COL10 038, 199AC, and 210), and one from South Shields (CS06 447) (Figure 4.32). In three of the occurrences, L5 and S1 were the affected vertebrae with the rotated facet on the left side twice and the right once. The other affected vertebrae were L1 and L2 on the left sides in COL10 210, and L4 and L5 again on the left sides in COL10 199AC.

Figure 4.32: Facet tropism in the fourth and fifth lumbar vertebrae of a male 30-34 years old. (**A**) Showing the left inferior apophyseal facet (arrow) rotated to face anteriorly on L4. (**B**) Showing the left superior apophyseal facet (arrow) rotated to face posteriorly on L5. (COL10 199AC, North Shields)

Rotated articular facets, when the rotations were symmetrical, occurred in two individuals from North Shields. Skeleton COL10 069 (9.5-10.5 years) showed rotation of both inferior apophyseal facets to face anteriorly on L3 and L4 while both superior apophyseal facets were rotated to face posteriorly on L4 and L5.

Skeleton COL10 123 had rotation of both inferior apophyseal facets to face anteriorly on L5 and the superior apophyseal facets to face posteriorly on S1.

4.3.14 Supernumerary Ribs

The data for cervical and lumbar ribs, special types of supernumerary ribs, were discussed in Sections 4.3.8.B and 4.3.8.C. The only other type of supernumerary rib seen was that associated with a supernumerary thoracic vertebra, but no intrathoracic ribs were recorded. All three individuals with thirteen thoracic vertebrae (see Section 4.3.9) had signs of thirteen sets of ribs. One individual, F119, a 30-34 year old male had bilateral costal facets on T13 and a minimum number of 25 ribs, as opposed to the normal 24. In the other two individuals, a male over 40 years old (F459) and a female over 45 years old (CS06 107), bilateral costal facets were seen on the body of T13 but no additional ribs were recovered.

4.3.15 Other Rib Anomalies

A broad range of congenital defects affecting the ribs was recorded but none in high numbers (Table 4.24). Two of the most commonly found rib anomalies, cervical and lumbar ribs, as well as supernumerary ribs, have already been discussed above (Sections 4.3.8.B, 4.3.8.C, and 4.3.14) along with other traits of border shifts. The next most common rib defect was the rudimentary 12th rib, which is similar to cervical and lumbar ribs in that it may be associated with a border shift. Eight individuals were affected, six of whom were individuals with border shifts at the thoracolumbar border. In these individuals, the twelfth thoracic vertebra had undergone partial lumbarization. There were rudimentary costal facets to match the rudimentary ribs. In the border shift associated occurrences, two were from Wharram Percy (both left side and unilateral), three were from North Shields (one bilateral, one right side unilateral with left unobservable, and one left side unilateral), and one was from South Shields (right side unilateral). The two occurrences of non-border shift associated rudimentary 12th ribs were associated

with normal T12 vertebrae. One individual was from Wharram Percy (left side unilateral) and the other from North Shields (left side unilateral).

Rib Anomalies	Present	Individuals	Prevalence
Bifid Rib	2	504	0.40%
Bridged Rib	1	504	0.20%
Broad Rib	2	504	0.40%
Cervical Rib	7	504	1.39%
Fused Rib	2	504	0.40%
Lumbar Rib	8	504	1.59%
Rib Spur	1	504	0.20%
Rudimentary Rib	8	504	1.59%
Supernumerary Rib	3	504	0.60%

Table 4.24: Rib anomalies present in the populations. Individuals were recorded as observable if at least one rib was present.

All other rib anomalies were recorded as occurring in only one or two individuals. Two occurrences of fused ribs were found at North Shields (Figure 4.33). The first individual, COL10 056, had the left 10^{th} and 11^{th} ribs fused starting about 3cm from the heads, but they were broken postmortem with only about 3cm of fused rib present; it was therefore not possible to determine how much of the rib was fused. This fusion may relate to the slightly displaced costal facet on T11 which was found on the transverse process in this individual. Radiography showed no signs of a traumatic origin. The second occurrence was based on a rib fragment from COL10 213. The fragment was potentially a lower left rib and had a bony bridge just lateral to the head protruding downwards and measuring 16mm wide. The heads and shafts of the ribs were broken off postmortem and could not be relocated with this individual.

Figure 4.33: Fused left ribs, possibly lower, in a male 25-34 years. (COL10 213, North Shields)

Both instances of bifid ribs were from North Shields (Figure 4.34). COL10 083 had a left rib that split at the sternal end for a length of 6.5mm. The rib was unusually broad and thin. The exact location of the rib in the body is unknown as it was not possible to piece together the rib beyond the bifid fragment. COL10 156 also had a bifid rib. The rib was from the left side, location unknown due to fragmentation, and had two sternal facets. As the individual was a non-adult around 3 years old, this condition may have become more pronounced as the individual grew if he or she had not passed away at a young age.

Figure 4.34: Bifid rib from the left with two sternal ends present in a non-adult around 3 years of age. Lines indicate the sternal ends while the arrow indicates the separation of the two. (COL10 156, North Shields)

The two occurrences of unusually broad ribs again both came from North Shields. Skeleton COL10 065 had bilateral broad ribs occurring at the 8th and 9th ribs on both sides. Both of the left ribs and the 8th right rib were one and a half times wider than adjacent ribs while the 9th right rib was twice as wide. For skeleton

COL10 131, only one broad rib occurred. This rib, from an unknown location on the right side, is visibly much broader than all the other ribs in the one quarter to one third of the shaft at the sternal end (Figure 2.29B).

Finally, there was one occurrence each of a rib spur, bridged ribs, and additional ribs. The rib spur was found in skeleton CN45 from Wharram Percy. The rib fragment, probably from the right side, had a small triangle of bone extending inferiorly for 5.0mm (Figure 2.29E). The spur was 5.0mm wide and was located 14mm from the sternal end. The bridged rib came from individual F360 of Fewston. The fragment exhibits bridging that was 11.4mm long and 11.3mm wide (Figure 2.29C). The external surface was smooth while the interior surface was uneven. The rib attached to the bony growth had been broken off and lost postmortem.

4.3.16 Pectus Carinatum

Pectus carinatum was observed in three individuals: F238 (?F, 30-34 years), CS06 107 (F, 45+ years), and CS06 559 (M, 40+ years) (Figure 4.35). In all three occurrences the sternum was bowed anteriorly/posteriorly. No rib changes or unusual ribs were seen in any of the individuals affected with pectus carinatum.

Figure 4.35: Pectus carinatum of the sternum in a possible female 30-34 years old. (F238, Fewston)

4.3.17 Os Acromiale

The presence of os acromiale at rural sites was found to be nearly statistically significant compared to the occurrence at urban sites (Figure 4.36, Tables 4.25 and 4.26), with a higher occurrence at the rural sites. When comparing

individual sites, data from Fewston was nearly statistically significantly different to those from North Shields, and the combined urban sites, with Fewston having higher rates of os acromiale. Data from North Shields was also nearly statistically significantly different from the combined rural sites which had a higher prevalence.

Figure 4.36: Os acromiale of the right scapula in an adult possible male. (**A**) Showing acromion with separate acromion process outlined in black. (**B**) Showing porous surface at abnormal joint. (CN02, Wharram Percy)

Site	Present	Observable	TPR
All Sites	19	327	5.81%
Fewston	4	34	11.76%
Wharram Percy	4	46	8.70%
North Shields	5	136	3.68%
South Shields	6	111	5.41%
Rural	8	80	10.00%
Urban	11	247	4.45%

Table 4.25: Presence of os acromiale out of all observable scapulae.

Site	vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	0.7170	0.0800	0.2445	-	0.0925
Wharram Percy	-	0.2334	0.4803	-	0.2668
North Shields	-	-	0.5491	0.0764	-
South Shields	-	-	-	0.2674	-
Rural	-	-	-	-	0.0945

Table 4.26: Statistical analysis (two tailed p values) for the presence of os acromiale. Green indicates a nearly significant result (p≤0.10).

Breaking down the data for os acromiale by laterality, there were no statistically significant differences when comparing unilateral versus bilateral or right versus left side. Occurrences of unilateral or bilateral os acromiale were only

recorded for individuals with both scapulae observable. When these features are compared by site there are no statistically significant results except for unilateral os acromiale. The high prevalence rate at Fewston proved to be nearly statistically significantly different from the urban sites, both individually and combined (Tables 4.27 and 4.28).

Site		Present	Observable	TPR	Side
All Sites	Bilat	2	113	1.77%	-
	Unilat	6	113	5.31%	(4x R, 2x L)
Fewston	Bilat	0	8	0%	-
	Unilat	2	8	25.00%	(1x R, 1x L)
Wharram Percy	Bilat	0	16	0%	-
	Unilat	1	16	6.25%	(1x R)
North Shields	Bilat	1	50	2.00%	-
	Unilat	2	50	4.00%	(1x R, 1x L)
South Shields	Bilat	1	39	2.56%	-
	Unilat	1	39	2.56%	(1x R)
Rural	Bilat	0	24	0%	-
	Unilat	3	24	12.50%	(2x R, 1x L)
Urban	Bilat	2	89	2.25%	-
	Unilat	3	89	3.37%	(2x R, 1x L)

Table 4.27: Presence of os acromiale by laterality. Individuals were only deemed observable if both scapulae could be examined for the trait.

Site	vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	0.2490	0.0876	0.0708	-	0.0528
Wharram Percy	-	1.0000	0.5010	-	0.4892
North Shields	-	-	1.0000	0.3211	-
South Shields	-	-	-	0.1504	-
Rural	-	-	-	-	0.1090

Table 4.28: Statistical analysis (two tailed p values) for the presence of unilateral os acromiale. Green indicates a nearly significant result (p≤0.10).

4.3.18 Aplasia of Ulnar Styloid Process

An aplastic ulnar styloid process was observed in three individuals. In skeleton CN02, an adult possible male, the right ulna demonstrated aplasia of the styloid process but the left ulna was missing post-mortem (Figure 2.33). A true unilateral occurrence was seen in skeleton CS06 062, a male 35-39 years old. He was lacking the styloid process on the left ulna while the right was normal. A

bilateral occurrence was found in skeleton CS06 998, a possible female over 45 years old.

4.3.19 Thanatophoric Dysplasia

Skeleton CS06 684 from South Shields was the only individual diagnosed with type 1 thanatophoric dysplasia (Figure 4.37). It was not possible to age this individual due to the abnormal long bones and lack of teeth but, due to the small size of the remains and the dysplasia being fatal *in utero* or shortly after birth, it was assumed the individual was a fœtus. The crude prevalence rates for this condition when looking at all ages of individuals were 0.15% (1/660) for all sites combined and 0.49% (1/204) for individuals from South Shields. The crude prevalence rates when looking at fœtal and neonate individuals only were 1.92% for all sites combined and 3.03% for individuals from South Shields. Widespread changes due to thanatophoric dysplasia were seen in these fragmentary remains. The femora were short and thick with broad metaphyses. They were slightly bowed and had a "telephone receiver" appearance. The tibiae and fibulae were also short and thick with broad metaphyses. The right humerus (left was missing post-mortem) was short and thick. The ulnae and left radius (right was missing post-mortem) were thickened but may represent a normal length. The hand bones (metacarpals?) displayed broadened metaphyses. The proximal ulnae and distal radius were particularly broad. Three lumbar vertebrae were prematurely fused at the pedicle, attaching the neural arches to the body. The vertebral bodies were slightly flattened (platyspondyly). The rib ends were flared. The ilia were thick and the acetabula were large. The only skull fragments present were the right side of the mandible and a portion of the right temporal bone which were both normal. This individual was diagnosed with type 1 of the condition due to the curved femora. In this type, a "cloverleaf skull" may or may not be present so the fact that this individual's cranium is largely absent does not affect the diagnosis.

Figure 4.37: Thanatophoric dysplasia in fœtal remains. (**A**) Showing all skeletal elements recovered during excavation of this individual. (**B**) Showing the comparison of long bones from the affected individual (right) and a normal fœtal individual (left; CS06 666, 26-30 weeks *in utero*). (**C**) Showing premature fusion of three lumbar vertebrae. (**D**) Showing the thickness of the ilia. (CS06 684, South Shields)

4.3.20 Charnel Deposits

Apart from the individual skeletons, charnel remains were examined from Fewston, North Shields, and South Shields for congenital defects. The Wharram Percy charnel remains were not available for study. In total, 14 individual defects were found from nine separate skeletons or contexts (Table 4.29). Defects observed were cleft neural arches, Klippel-Feil syndrome, transitional vertebrae, a rudimentary apophyseal facet, a lumbar or rudimentary rib, and aplastic transverse processes (Figure 4.38).

Skeleton	Defect	Location	Description
F278C	cleft neural arch	C7	-
	Klippel-Feil Syndrome	unknown cervical vertebrae	non-adult vertebra - inferior apophyseal facet present but flattened, superior apophyseal facet absent or displaced and missing post-mortem
F279C	transitional vertebra?	L1?	superior apophyseal facets face posterior, inferior apophyseal facets face lateral, no costal facets present
F305C	cleft neural arch	two unknown cervical vertebrae	both non-adult vertebrae - both cleft just right of midline
		upper thoracic	non-adult vertebra - cleft just right of midline
	rudimentary apophyseal facet	unknown cervical vertebra	non-adult vertebra - right superior apophyseal facet rudimentary and convex, left side is normal
COL10 155C	lumbar rib or rudimentary rib	-	only one lumbar vertebra present, no costal facets seen, no thoracic vertebrae present
COL10 189C	transitional vertebra	T12	T11 - both inferior apophyseal facets face lateral, T12 - both superior apophyseal facets face medial
CS06 232	transitional vertebra	T12	T12 - superior apophyseal facets face medial, left costal facet roughly half the size of the right, other facets normal, T11 not present post-mortem
CS06 363	transitional vertebra	T12	T11 - both inferior apophyseal facets face lateral, T12 - both superior apophyseal facets face medial, all other facets normal
CS06 462	aplastic transverse processes	T12	absent transverse processes, costal facets in location where process should have been
	transitional vertebrae	L1	both superior apophyseal facets facet posterior, aplastic right transverse process, rudimentary costal facet in place where process should have been
		L5	complete sacralization, L1-L5 present

Table 4.29: All congenital defects found in charnel material from Fewston, North Shields, and South Shields. Charnel from Wharram Percy was not available for study.

Skeleton	Defect	Location	Description
CS06 527DC	transitional vertebra	T12	right superior apophyseal facet faces medial, left faces posterior, inferior facets normal, unobservable for costal facets
CS06 1000C	transitional vertebra	T12	left superior apophyseal faces medial, right faces posterior, all other facets normal

Table 4.29 (continued): All congenital defects found in charnel material from Fewston, North Shields, and South Shields. Charnel from Wharram Percy was not available for study.

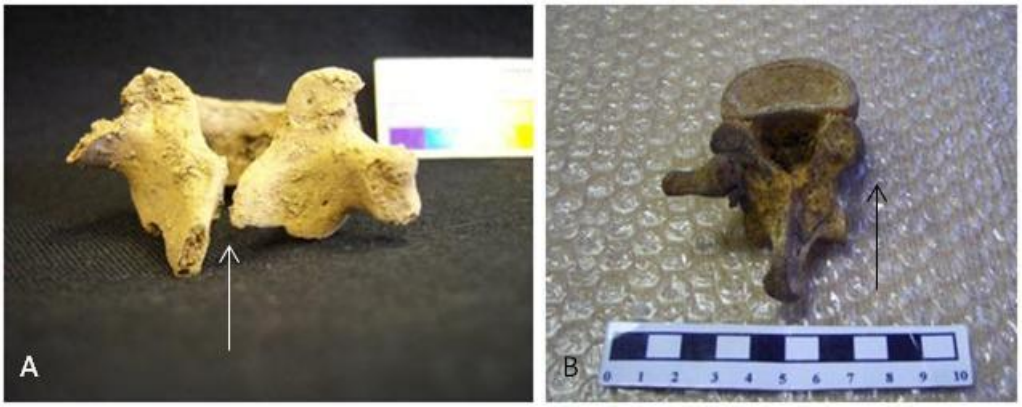

Figure 4.38: (**A**) A cleft neural arch in a thoracic vertebra from a non-adult. (F305C, Fewston) (**B**) Aplastic right transverse process in a transitional first lumbar vertebra. (CS06 462, South Shields)

When the charnel remains information for cleft neural arch and transitional vertebrae was added into the overall data, changes to the pattern of statistical difference were only seen for transitional vertebrae at the thoracolumbar border in the caudal (lumbar ribs) direction. As seen in Table 4.30, the changes occurred in reference to Fewston in individuals with T11, T12, T13, or L1 present. Fewston had statistically significantly more shifts than North Shields (previously nearly statistically significant) and nearly statistically significantly more than South Shields (previously, no significant result).

Site		vs F	vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	W/ Charnel	-	0.1184	0.2250	0.0930	-	0.0229
	Without	-	0.2067	0.0558	0.1374	-	0.0476
Wharram Percy	W/Charnel	-	-	1.0000	1.0000	-	1.0000
	Without	-	-	1.0000	1.0000	-	1.0000
North Shields	W/ Charnel	-	-	-	0.7448	0.1270	-
	Without	-	-	-	1.0000	0.2160	-
South Shields	W/ Charnel	-	-	-	-	0.3802	-
	Without	-	-	-	-	0.5167	-
Rural	W/ Charnel	-	-	-	-	-	0.1490
	Without	-	-	-	-	-	0.2253

Table 4.30: Statistical analysis (two tailed p values) for the caudal shifts at the thoracolumbar border for individuals with a T11, T12, T13, or L1 present, comparing the data with and without charnel. Yellow indicates a significant result (p≤0.05) while green indicates a nearly significant result (p≤0.10).

4.4 "Stress" Indicator Frequency

For all "stress" indicators combined (porotic hyperostosis, cribra orbitalia, dental enamel hypoplasia, and tibial and fibular periosteal new bone formation), all sites had similar prevalence rates ranging between 52.55 and 56.90% (Table 4.31). Prevalence rates were determined according to the number of individuals that could be examined for at least one "stress" indicator (*i.e.* at least one orbit, skull fragment, or tooth, or one half of a tibia or fibula present and preserved well enough for macroscopic observation). None of the differences between the sites proved to be statistically significant. Below, the individual indicators of porotic hyperostosis, cribra orbitalia, dental enamel hypoplasia, periosteal new bone formation of the tibia and fibula, and stature are explored further.

Site	Present	Observable	CPR
All Sites	341	626	54.47%
Fewston	72	137	52.55%
Wharram Percy	33	58	56.90%
North Shields	134	238	56.30%
South Shields	102	193	52.85%
Rural	105	195	53.85%
Urban	236	431	54.76%

Table 4.31: The presence of "stress" indicators at all sites. "Stress" indicators analysed were porotic hyperostosis, cribra orbitalia, dental enamel hypoplasia, and tibial and fibular periosteal new bone formation. Observable denotes the number of individuals where at least one "stress" indicator could be observed.

4.4.1 Porotic Hyperostosis

Eight potential occurrences of porotic hyperostosis (PH) were found in the sample (Figure 4.39, Table 4.32). The occurrences were distributed as follows: one individual from Fewston, one from Wharram Percy, and six from North Shields. The only comparison found to be statistically significant was between North and South Shields, with North Shields having significantly more individuals affected (p=0.0393).

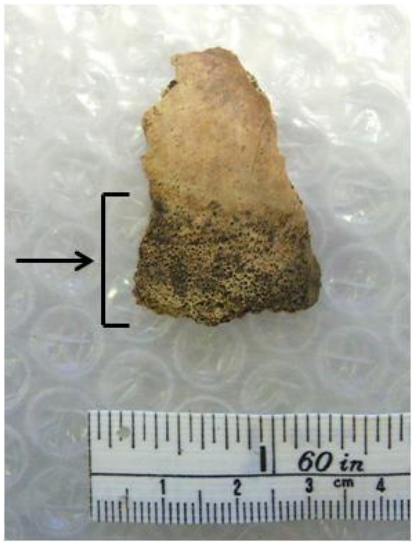

Figure 4.39: Porotic hyperostosis on a parietal bone fragment from a non-adult less than 1 year old. The area of porosity is marked by the arrow. (COL10 003, North Shields)

Site	Present	Observable	TPR
All Sites	8	528	1.52%
Fewston	1	107	0.93%
Wharram Percy	1	44	2.27%
North Shields	6	215	2.79%
South Shields	0	162	0%
Rural	2	151	1.32%
Urban	6	377	1.59%

Table 4.32: Presence of porotic hyperostosis for all sites. Observable individuals had at least one skull fragment present.

4.4.2 Cribra Orbitalia

Prevalence rates for cribra orbitalia (CO) were calculated according to both the numbers of individuals and number of orbits observable (Figure 4.40, Table 4.33). Both recording systems produced similar patterns when looking at the data (Table 4.34). When comparing orbits, the frequency at Fewston was statistically significantly greater than at any other site and the individuals had statistically more occurrences than all sites, except Wharram Percy, when comparing individuals. For both orbits and individuals, both North and South Shields had a statistically lower rate than the combined rural sites. The combined urban sites also had a statistically lower rate for both orbits and individuals than the combined rural sites.

Figure 4.40: Bilateral cribra orbitalia in a possible male 30-34 years old. The areas of porosity are marked by the arrows. (COL10 112, North Shields)

Site	Present	Individuals	Orbits	TPR
All Sites	92	461	-	19.96%
	138	-	831	16.61%
Fewston	26	74	-	35.14%
	39	-	125	31.20%
Wharram Percy	9	44	-	20.45%
	14	-	84	16.67%
North Shields	37	196	-	18.88%
	53	-	355	14.93%
South Shields	20	147	-	13.61%
	32	-	267	11.99%
Rural	35	118	-	29.66%
	53	-	209	25.36%
Urban	57	343	-	16.62%
	85	-	622	13.67%

Table 4.33: Presence of cribra orbitalia as recorded for both individuals and by orbit.

Site		vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	Individ	0.1006	0.0062	0.0004	-	0.0006
	Orbit	0.0228	0.0002	0.0001	-	0.0001
Wharram Percy	Individ	-	0.8330	0.3370	-	0.5248
	Orbit	-	0.7359	0.2700	-	0.5022
North Shields	Individ	-	-	0.2410	0.0372	-
	Orbit	-	-	0.3455	0.0026	-
South Shields	Individ	-	-	-	0.0021	-
	Orbit	-	-	-	0.0002	-
Rural	Individ	-	-	-	-	0.0032
	Orbit	-	-	-	-	0.0002

Table 4.34: Statistical analysis (two tailed p values) of the presence of cribra orbitalia as recorded both by individual and by orbit. Yellow indicates a statistically significant result ($p \leq 0.05$).

Using individuals where both orbits were observable for cribra orbitalia, bilateral involvement was more common than unilateral (Table 4.35). There were 46 instances of bilateral CO but only 28 occurrences of unilateral (split evenly with fourteen occurrences each for right and left). Statistical significance for bilateral occurrence has the same pattern as occurrence in individuals, with Fewston and the combined rural sites having more occurrences than their urban counterparts (Table 4.36). Similar patterns are seen when comparing the unilateral occurrences but with less significance.

Site		Present	Observable	TPR
All Sites	Bilat	46	370	12.43%
	R Only	14	370	3.78%
	L Only	14	370	3.78%
Fewston	Bilat	13	51	25.49%
	R Only	4	51	7.84%
	L Only	1	51	1.96%
Wharram Percy	Bilat	5	40	12.50%
	R Only	3	40	7.50%
	L Only	0	40	0%
North Shields	Bilat	16	159	10.06%
	R Only	3	159	1.89%
	L Only	10	159	6.29%
South Shields	Bilat	12	120	10.00%
	R Only	4	120	3.33%
	L Only	3	120	2.50%
Rural	Bilat	18	91	19.78%
	R Only	7	91	7.69%
	L Only	1	91	1.10%
Urban	Bilat	28	279	10.04%
	R Only	7	279	2.51%
	L Only	13	279	4.66%

Table 4.35: Location of cribra orbitalia in individuals with both orbits observable.

Site		vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	Bilat	0.1847	0.0093	0.0162	-	0.0046
	R Only	1.0000	0.0609	0.2407	-	0.0727
	L Only	1.0000	0.3023	1.0000	-	0.7043
Wharram Percy	Bilat	-	0.7731	0.7672	-	0.5838
	R Only	-	0.0970	0.3678	-	0.1173
	L Only	-	0.2171	0.5737	-	0.3834
North Shields	Bilat	-	-	1.0000	0.0360	-
	R Only	-	-	0.4680	0.0395	-
	L Only	-	-	0.1616	0.0606	-
South Shields	Bilat	-	-	-	0.0488	-
	R Only	-	-	-	0.2133	-
	L Only	-	-	-	0.6357	-
Rural	Bilat	-	-	-	-	0.0179
	R Only	-	-	-	-	0.0501
	L Only	-	-	-	-	0.2029

Table 4.36: Statistical analysis (two tailed p values) for location of cribra orbitalia for all individuals with both orbits observable. Yellow indicates a significant result ($p \leq 0.05$) while green indicates a nearly significant result ($p \leq 0.10$).

4.4.3 Dental Enamel Hypoplasia

Two hundred and twenty four individuals out of 471 were found to have dental enamel hypoplasia (DEH) on at least one tooth (Figure 4.41, Table 4.37). Individuals were observable if they had a least one tooth crown present. Fifty individuals from Fewston, 20 from Wharram Percy, 97 from North Shields, and 77 from South Shields all had a DEH. When the sites were compared, no statistically significantly different results were found.

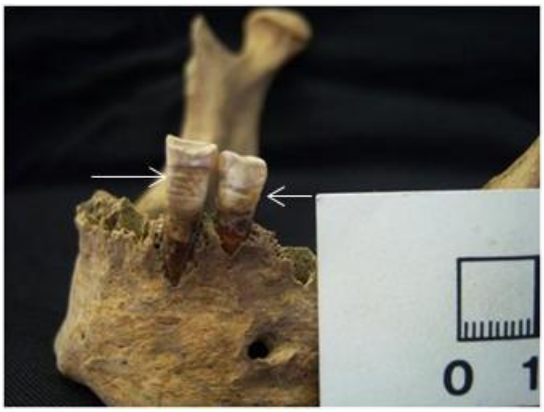

Figure 4.41: Dental enamel hypoplasia on the mandibular left canine and first premolar of a 40-44 years old possible female. (COL10 225, North Shields)

Site	Present	Individuals	TPR
All Sites	244	471	51.80%
Fewston	50	104	48.08%
Wharram Percy	20	37	54.05%
North Shields	97	194	50.00%
South Shields	77	136	56.62%
Rural	70	141	49.65%
Urban	174	330	52.73%

Table 4.37: Presence of dental enamel hypoplasia. Individuals were observable if at least one tooth crown was present.

4.4.4 Periosteal New Bone Formation

Periosteal new bone formation (PNBF) was recorded for both the tibiae and fibulae (Figure 4.42, Tables 4.38 and 4.39). This type of new bone formation occurred overall more often in the tibia (15.51%) than in the fibula (12.12%). For the tibia, there were nearly statistically more occurrences in individuals at the combined urban sites than at Fewston (p=0.0816) and, again, nearly statistically

significantly more at South Shields than at the combined rural sites (p=0.0673). For all comparisons for the fibula, no statistically significantly different results were found.

Figure 4.42: Woven periosteal new bone formation on the right tibia of an adult of unknown age and sex. (CS06 910, South Shields)

Site	Present	Individuals	TPR
All Sites	74	477	15.51%
Fewston	10	87	11.49%
Wharram Percy	5	48	10.42%
North Shields	31	199	15.58%
South Shields	28	143	19.58%
Rural	15	135	11.11%
Urban	59	342	17.25%

Table 4.38: Presence of tibial periosteal new bone formation for all sites. Individuals were deemed observable if at least one half of one tibia was present.

Site	Present	Individuals	TPR
All Sites	52	429	12.12%
Fewston	8	68	11.76%
Wharram Percy	6	44	13.64%
North Shields	21	186	11.29%
South Shields	17	131	12.98%
Rural	14	112	12.50%
Urban	38	317	11.99%

Table 4.39: Presence of fibular periosteal new bone formation for all sites. Individuals were deemed observable if at least one half of one fibula was present.

4.4.5 Stature

The average heights for people at each site can be found in Table 4.40 and Figure 4.43. The tallest average male height was found at Fewston (176.43cm/

69.46in) and the shortest at Wharram Percy (169.65cm/66.79in). For the females, the tallest average height was found at North Shields (160.79cm/63.3in) and the shortest at Fewston (158.99cm/62.59in). Fewston males were statistically taller than males from any other site or the urban sites combined. When comparing between sites for the females, no statistically significant results were found.

Site	Sex	Centimetres	Inches	Individuals
Fewston	MALES	176.43	69.46	17
	FEMALES	158.99	62.59	15
Wharram Percy	MALES	169.65	66.79	24
	FEMALES	160.34	63.13	17
North Shields	MALES	170.56	67.15	63
	FEMALES	160.79	63.30	36
South Shields	MALES	171.80	67.64	53
	FEMALES	160.06	63.01	41
Rural	MALES	172.46	67.90	41
	FEMALES	159.71	62.88	32
Urban	MALES	171.13	67.37	116
	FEMALES	160.40	63.15	77

Table 4.40: Mean stature for all adult individuals for whom sex could be determined and had at least one intact long bone suitable for measuring.

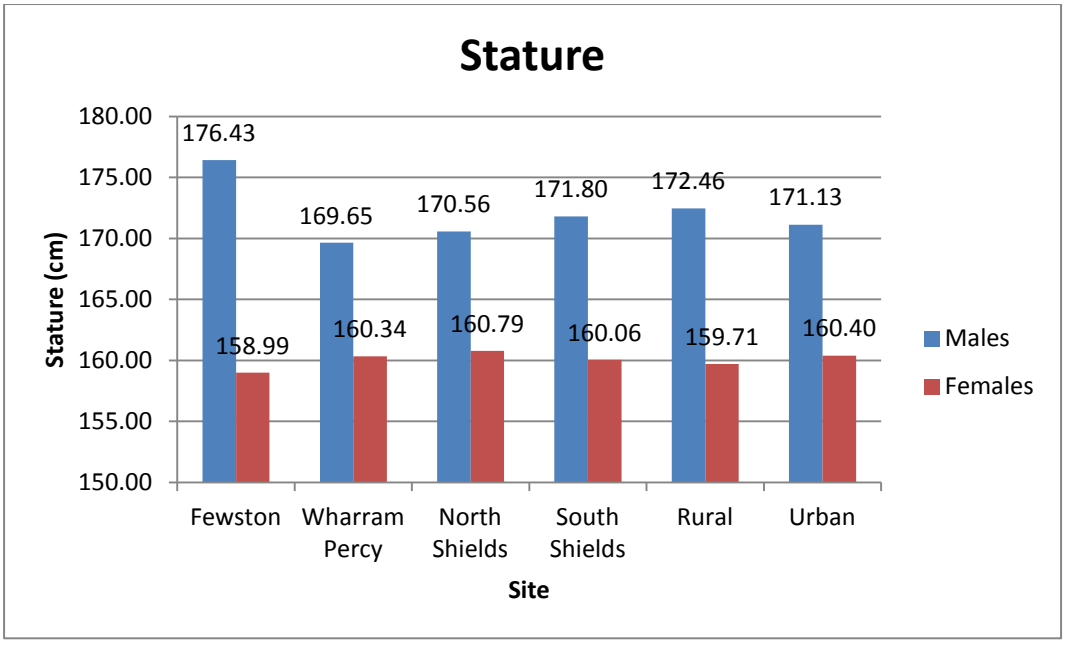

Figure 4.43: Figure showing the mean stature for all adults for whom sex could be determined and had at least one intact long bone suitable for measuring.

4.5 Conclusions

This chapter has detailed the results gained through the bioarchaeological analysis of 660 individuals and multiple charnel deposits. A variety of congenital defects affecting most regions of the skeleton were found during this study. When the presence of all congenital defects were combined, the difference between rural and urban sites was not significant, but when the individuals either under eighteen years or four years of age were removed from the picture, the data became statistically significant (Section 4.3.1). Wharram Percy had the highest prevalence rate (CPR) overall for congenital defects while Fewston had the lowest. "Stress" indicators were also found amongst the study populations. When the presence of all "stress" indicators were combined, the difference between rural and urban sites was not found to be statistically significant. Again, Wharram Percy had the highest prevalence rate (CPR) overall for "stress" indicators while Fewston had the lowest. In the next chapter, the results reported here will be put into context with reference to the research on teratogens and living conditions presented in Sections 2.2 and 2.3. Additionally, the data presented here will be compared with contemporaneous sites to look for any similarities or differences.

CHAPTER 5
DISCUSSION

'Skeletal lesions have little comparative value if studied in isolation; their context is essential for exploratory analysis of trends and patterns in health.'
(Steckel, 2007:17)

5.1 Introduction

Findings by Sture (2001) and Kase (2010) on the medieval and post-medieval periods in Britain have shown that higher frequencies of congenital defects were present in populations from urban areas and that this may be linked to poor living conditions in urban environments. Building on from these studies, as detailed in Chapter 1, the hypothesis investigated by this study was that, in Northeast England, congenital defects would be more common in urban populations than in rural ones, due to the poorer living conditions associated with urbanised, industrial post-medieval towns. For this reason, congenital defects and "stress" indicators were recorded in four skeletal populations, two from urban and two from rural contexts. However, the results of this bioarchaeological recording, as detailed in Chapter 4, was unable to support the hypothesis as there was no statistical difference between urban and rural sites for the presence of congenital defects.

This chapter will discuss the potential reasons why the hypothesis had to be rejected. First, the main congenital defects findings from Chapter 4 are summarised in Section 5.2.1. The patterns seen in these results are then explored, alongside documentary evidence of living conditions at the study sites and clinical information on teratogens, to evaluate the impact of living conditions, migration, socioeconomic status, and the aetiology of congenital defects on the prevalence of congenital defects in the past. The examination of the aetiology of congenital defects also assesses the suitability of congenital defects as indicators of past living conditions (Section 5.2). Next, Section 5.3 looks at the "stress" indicator results and

discusses these in relation to the patterns seen in congenital defects. Additionally, the effect of living conditions on the frequencies of "stress" indicators is explored.

Section 5.4 compares the data collected in this study with contemporaneous populations to explore how typical the study data are for the period. Urban sites used for comparison include St Martin's from Birmingham, St Peter's from Barton-upon-Humber, and the sites of Cross Bones, Chelsea Old Church, and St George's Church from London. Three Quaker burial grounds located at Kingston-upon-Thames, King's Lynn, and St Ives, Cambridgeshire are compared to the Quaker burial ground of North Shields. Additionally, Wharram Percy is examined for changes in congenital defect frequency through time using data from Sture's (2001) research on individuals from the late medieval period at that site.

The urban/rural divide is then examined in Section 5.5 to determine the validity of this separation in bioarchaeological studies of the 18th and 19th centuries. This section returns to the definitions of urban and rural provided in Chapter 1 and what this means for the design of this study. Finally, the limitations inherent in bioarchaeological studies such as this one are discussed with reference to how these may have affected the data and its interpretation.

5.2 Congenital Defects

As noted above, the hypothesis that congenital defects would be more prevalent in urban populations cannot be supported. The reasons examined in this section are (i) the similar levels of detrimental effects in the living conditions at both urban and rural sites, (ii) migration between urban and rural locations artificially raising or lowering the observed prevalence rates, (iii) the effect of socioeconomic status on the occurrence of congenital defects, and (iv) the inability to remove genetic causation from studies such as this. However, before moving onto the discussion of these factors, relevant results from Chapter 4 are summarised showing the lack of statistically significant results between urban and rural sites.

5.2.1 Results Summary

As seen in Chapter 4, prevalence rates for combined congenital defects were not statistically different for people buried at the combined urban sites compared to combined rural sites (p=0.0846). When comparing individual sites for combined congenital defects, people buried at Fewston had statistically significantly fewer occurrences of defects than any other site, or the urban sites combined. The other rural site was at the other end of the spectrum. Statistically significant results were found between the individuals from Wharram Percy and those from Fewston, South Shields, and the combined urban sites, with nearly statistically significant results compared to individuals from North Shields (significant at 90%). In all comparisons, Wharram Percy had the higher prevalence rates. The differences between the urban sites were not statistically significant, with North Shields having only a slightly higher prevalence rate than South Shields. This created a pattern of individuals from Wharram Percy (43.33%) having the highest prevalence rates followed by North Shields (33.47%), South Shields (30.88%), and then Fewston (18.54%).

Statistical tests were also undertaken restricting the populations to individuals over four years old and individuals over 18 years old. These ages were chosen as there is the inability to record some congenital defects in younger individuals whose bones have not finished fusing (growth) yet. Most bones have fused by the age of 18 years, while the spine has finished fusing by the age of 4 years (Scheuer and Black, 2000). Prevalence rates for combined congenital defects were statistically different with higher rates for people buried at the combined urban sites compared to those from the rural sites, for both age restricted tests. While this could be considered sufficient evidence to reject the null hypothesis, the results should be examined further. Although there is a significant difference between urban and rural site frequency, the ordering of the sites in terms of prevalence rates remained virtually the same as that for all individuals. The data from Wharram Percy still showed the highest rates (51.02% adults, 47.27% >4), followed by South Shields (45.31% adults, 42.47% >4), North Shields (41.98% adults, 41.80% >4), and finally Fewston (25.00% adults, 20.14% >4). This raises the

question of whether we can definitively say that urban sites had a higher prevalence rate when the highest rate was at a rural site. The two urban sites had reasonably similar rates throughout the analysis of congenital defects (for individual and combined defects), but at times the two rural sites were very different. This implies that the urban sites are potentially representative or "normal" for urban locations of the time. It also implies that one of Wharram Percy or Fewston is atypical of rural sites for the period, or indeed that both are. The stark contrast between the two rural sites means that they cannot both be what is expected of rural locations: either there is little to no difference between urban and rural sites of the period (meaning Fewston is an anomaly), or there is a difference between urban and rural sites (making Wharram Percy unusual). In short, the data for the combined congenital defects for the entire population and for those in age restricted categories do not show a statistically significant difference between the urban and rural sites that can be interpreted in terms of an urban/rural divide.

Looking at the individual defects, the conundrum continues. For individual defects, individuals from Fewston generally had the lowest rates, but no consistent pattern was seen for the other sites. Only two defects produced statistically significant or nearly significant results between sites: os acromiale and thoracolumbar border shifts. Os acromiale was more common in individuals from rural sites, and this difference was nearly statistically significant ($p=0.0945$, significant at 90%). The aetiology of this defect was originally attributed to an unhealed fracture of the acromion and as such was activity induced. Now it is more commonly ascribed to a congenital origin (see Section 2.4.16 for further discussion). As such, differential activities/occupations occurring at the two types of site can be ruled out as a cause. Unlike many of the defects recorded in this study, os acromiale takes on a present or absent appearance - either the defect is there for each scapula or it is not, much like a non-metric trait. By contrast, most congenital defects have varying expressions in appearance, for example a cervical rib may be a slight protruding nodule or a fully formed separate rib with all the normal rib anatomy in place. This may indicate that the presence of os acromiale may be affected more by genetics as a non-metric trait, than environmental factors.

However, to the author's knowledge, little clinical or bioarchaeological research has been undertaken to examine the effect of genetics on the formation of os acromiale (see Case *et al.*, 2006).

The second defect that gave statistically significant results was border shifts at the thoracolumbar border. For shifts in combined caudal and cranial directions, the results were nearly statistically significant for the sites (p=0.0865, significant at 90%). This became significant when only those individuals with complete spines were examined for the defect (p=0.0402). For lumbarization of T12 (cranial shifts), the results showed that individuals from urban sites had a statistically significantly higher prevalence of shifts than those from rural sites (p=0.0242). Unlike os acromiale, there are varying stages of shifting, perhaps implying this could be more controlled by environmental factors. However, as this is the only defect to statistically follow the hypothesis that urban individuals will be more greatly affected by defects, this cannot be used to conclusively state that urban environments led to defects.

5.2.2 Living Conditions

Considering both combined and individual congenital defects, there was no conclusive proof that individuals buried in urban environments had statistically significantly more defects than those buried in rural contexts (see Chapter 4 and Section 5.2.1). The first possibility for the lack of an urban/rural divide in congenital defects is that urban and rural living conditions of the 18th and 19th centuries were not actually all that different. Section 2.3 demonstrated that living in rural environments had the potential to be detrimental to health with both urban and rural contexts containing risk factors for the health of individuals, even when *in utero*. The living conditions of all four study sites were discussed in detail in Section 2.3 but will be mentioned here again with reference to specific conditions that are known to be teratogenic (*i.e.* causing congenital defects).

Housing conditions were dependent on both the time period and the income of the inhabitants. For example, the Hearth Tax returns for County Durham

1666, which included South Shields, demonstrated a worsening of housing conditions for most of the poor and wage-labourers of the area (Green and Parkinson, 2006). However, skilled labourers, craftsmen, and farmers with enough land holdings to benefit from the agricultural market did experience improvements in housing (*ibid.*). By the nineteenth century, in the urban environments, entire families were housed in one or two rooms with few windows (Reid, 1845a, b; Robinson, 1847; Woodward, 1995). Houses would be subdivided to provide accommodation for multiple families and crammed together on narrow alleyways (Atkinson, 1989; Hodgson, 1903; Reid, 1845a, b; Woodward, 1995). In rural areas, families were similarly living in two or three rooms with few windows (Giles, 2006; Hey, 1986; Jennings, 1967; Mercer, 1975; Neave and Neave, 2006). The number of individuals per square footage of living space was likely to have actually been relatively similar between urban and rural settlements although no hard figures for the rural sites have been found to support this. The major difference between the two areas would have been population density. An urban family would have been surrounded on all sides by other families while the rural tenants would have had much more space (Berry, 2010; Garson, 1992; Grundy *et al*., 1992; Reid, 1845a, b). They would have exchanged the closeness of humans for the closeness of livestock. While urban dwellers would have been exposed to infectious disease due to the close living quarters with other humans, the rural dwellers would have been exposed to diseases from sharing close quarters with animals, in addition to interacting with other humans within their settlement and further afield at markets (Alred, 1997; Harker, 1988; Hey, 1986). However, in the lower population densities of rural communities, it is expected there was more opportunity for avoiding the closeness of other humans. Close living arrangements, with other humans or with livestock, would have contributed to the spread of infectious disease some of which are known to be teratogenic. High fevers brought on by influenza and other infections, rubella, and lymphocytic choriomeningitis virus are known to detrimentally affect the developing skeleton *in utero*, resulting in cleft lip and palate, microphthalmia, facial defects, microcephaly, macrocephaly, hydrocephalus,

and neural tube defects (Barton and Mets, 2001; Connor and Ferguson-Smith, 1997; Czeizel *et al.*, 2008; Hannachi *et al.*, 2009; Lancaster, 2011; Rousseau *et al.*, 1997).

Both urban and rural settlements lacked any sort of proper sanitation or disposal of waste (Addyman, 1989; Hodgson, 1903; Hood Coulthard, 1959, 1960; Jennings, 1967; Reid, 1845a, b; Simpson, 1988; Southern, 2003). Here again, the main teratogenic concern would have been the spread of infectious disease through humans and animal fæces. Additionally, the squalor of these urban areas would have attracted vermin such as rats which are carriers of lymphocytic choriomeningitis virus, a known teratogen (Barton and Mets, 2001). Rural dwellers may have been shielded from much of the ill effects caused by standing effluvia due to the space inherent in villages but may have been exposed to night soil (human excrement) for fertilization of their fields. Furthermore, the effluvia inherent in both settlement types could have easily contaminated the drinking water supplies (Alred, 1997; Jennings, 1967; Harker, 1988; Hey, 1986; Hodgson, 1903; Reid, 1845b). Textile production in the rural areas would also have released soap, tallow, dyes, and oils into waterways polluting drinking water (Jennings, 1967; Singleton, 1970).

Pollution, particularly coal smoke, was one of the most common features associated with urban areas of the post-medieval period. Although more of a problem in urban areas, rural areas were not entirely immune from it. Many industries were located in North and South Shields that relied heavily on burning coal including glass works, salt making, and chemical works (Hodgson, 1903; Linsley, 1992; Reid, 1845b; Thornborrow, 1968, 1971a, 1988). Additionally, pit-engines, coal powered ships, and prevailing westerly winds from Newcastle-upon-Tyne all contributed to the coal smoke pall of the towns (Hodgson, 1903; Hood Coulthard, 1960; Moffat and Rosie, 2005; Reid, 1845a, b). In the rural areas of North Yorkshire, agricultural and textile mills could produce copious quantities of coal smoke (Alred, 1997). Domestically, coal was a common fuel in both the urban and rural study sites, meaning indoor environments would not have been protected from the coal smoke due to the inefficient chimneys of the period (Harker, 1988; Hey, 1986;

Jennings, 1967; Neave and Neave, 2006; Reid, 1845a, b). There were no statistically significant differences between individuals from North Shields and Fewston for chronic maxillary, sphenoid, and frontal sinusitis, and inflammatory rib lesions, all of which are attributed to exposure to coal smoke (Roderick, 2011). Mercury and arsenic are components of ever present coal smoke, at both the urban and rural study sites, and can lead to cleft lip and palate, encephaly, neural tube defects, fused vertebrae, occipitalization, fused ribs, clubbed feet, and syndactyly (Hill *et al.*, 2008; Léonard and Lauwerys, 1980; Machado *et al.*, 1999; Tchounwou *et al.*, 2003). Coal fires also created environments rich with carbon monoxide and carbon dioxide, creating hypoxic environments and producing skeletal defects such as cleft lip, microcephaly, craniofacial defects, skull defects, mandible defects, neural tube defects, wedged vertebrae, fused vertebrae, absent vertebrae, hemivertebrae, scoliosis, fused ribs, lumbar ribs, limb reduction defects, and ectrodactyly (Ema *et al.*, 2010; Loder *et al.*, 2000; Ornoy *et al.*, 2010; Singh *et al.*, 1993; Webster and Abela, 2007).

The chemical works not only produced coal smoke but also hazardous gases such as hydrochloric acid and carbon dioxide (Atkinson, 1989; Campbell, 1968; Moffat and Rosie, 2005; Warren, 1980). Lead and sulphur dioxide were released into the atmosphere by lead ore smelting (Atkinson, 1989; Jennings, 1967), while selenium was released into the water system by the coal mining which took place at South Shields and in North Yorkshire, the latter is known to lead to spinal curvatures (de Rosemond *et al.*, 2005; Flohé, 2009; Miller *et al.*, 2009). Lead, released into the atmosphere during lead ore smelting, produces fused cervical vertebrae and delayed ossification of bones (Jacquet and Gerber, 1979). Indeed, elevated lead levels have been found in the tooth enamel of individuals from the Quaker burial ground, North Shields, indicating that at least that population was exposed to lead, probably due to the industry of the region (Ostrander, 2013).

The staple diet of both rural and urban dwellers was quite similar and consisted of oatmeal, tea, milk, butter, potatoes, bread, and meat when it could be afforded (Drummond and Wilbraham, 1957; Hey, 1986; Ketabgian, 2007; Wilson,

2003). Foodstuffs available in towns were generally of poorer quality and less fresh than that which could be acquired in rural areas, especially when looking at working-class meals (Drummond and Wilbraham, 1957; Freeman, 1989; Hey, 1986). Food and drink in towns was commonly adulterated with a wide variety of items including lead used to sweeten foodstuffs (Drummond and Wilbraham, 1957; Freeman, 1989; Gray, 2009; Ketabgian, 2007; Wilson, 2003). Consumption of vegetables and fruit was limited in towns due to their inability to grow these items themselves (Drummond and Wilbraham, 1957; Freeman, 1989; Thirsk, 2006), whereas rural dwellers would have had the ability to grow their own fresh foods or scavenge them from common lands (Hey, 1986; Jennings, 1967; Thirsk, 2006; Wilson, 2003; Woodward, 1995). Adequate nutrition in the mothers-to-be of the past is the concern here for teratogenic effects. A lack of zinc, commonly seen in individuals with limited meat in their diet, can produce cleft lip and palate, anencephaly, neural tube defects, spina bifida, absent vertebrae, fused thoracic and lumbar vertebrae, spinal curvatures, rudimentary ribs, fused ribs, syndactyly, clubbed feet, and agenesis of limbs (Hurley, 1981; Shah and Sachdev, 2001; Uriu-Adams and Keen, 2010). Too much vitamin A, gained by eating excess liver, carrots, broccoli, and spinach, can lead to craniosynostosis, calvarium hypoplasia, mandible malformations, cleft palate, craniofacial defects, supernumerary vertebrae, absent vertebrae, and premature long bone fusion (Abbott, 2010; Chapman, 2012; Laue *et al.*, 2011; Villenueve *et al.*, 2006).

As can be seen above and in Sections 2.2 and 2.3, known teratogenic conditions existed at all four study sites in the 18th and 19th centuries, regardless of whether the locations were classified as urban or rural. While not every risk factor was found in both contexts, a variety of these harmful conditions can be found in both urban and rural settlements. In short, urban and rural sites were the same in different ways. This may account for the lack of statistical differences between prevalence rates of congenital defects at urban and rural locations.

5.2.3 Migration

A second potential reason for the lack of statistical significance in the differences of congenital defects found in urban and rural sites is migration. During the 18th and 19th centuries, there was an increase in migration, especially of young adults, into urban centres in search of employment in the newly founded industries (Alderman, 1986; Atkinson, 1989; Burnett, 1978; Green, 2003; Morris and Rodger, 1993; Sharpe, 2000; Woodward, 1995; Wrigley, 2004). This poses a conundrum. While it can be easily assumed that every individual buried in the cemeteries at North and South Shields died in those towns, there is no way of knowing whether or not they were born there. As the hypothesis for this thesis is that living conditions were better in rural areas and that individuals from there would have lower rates of congenital defects, then these unaffected (rural) individuals moving into towns would lower the prevalence rates for the population. Looking at the parish registers for St Hilda's in South Shields, many of the individuals having their children baptised had been born themselves in and around South Shields. However, there were entries showing individuals born in locations across Britain and even from other countries (Table 5.1) (MF831; PR/WP 3). This contrasts sharply with the same records for Fewston and Wharram Percy which shows that the majority of the parents of children being baptised came from within the parish (PR/FEW 1/16, 1/17; PR/WP 3). North and South Shields, being seaports, had mariners from across Britain and further afield finding themselves in the towns due to employment opportunities, or even just passing through on their trade routes (Charlton, 2008; Garson, 1992; Hodgson, 1903; Hood Coulthard, 1960; Jackson, 2000; Sharpe, 2000; Simpson, 1988). Any of these individuals could have affected the frequency rates in this study if they had been born in healthier regions only to pass away amongst the poorer living conditions of North or South Shields (see Sections 2.3 and 5.2.2).

Nativity	Individuals
South Shields and Immediate Area	**180**
South Shields	155
Cullercoates	1
Howdon Pans	1
Jarrow	8
North Shields	9
Shields	1
Westoe	5
Northumberland	**18**
Alnmouth	1
Alnwick	2
Bamburgh	1
Bedlington	1
Berwick	1
Hexham	3
Morpeth	5
Newbiggin, Northumberland	1
Northumberland	3
Yorkshire	**24**
Little Horton	1
Madley, Yorkshire	1
Reeth, Co. York	1
Rounton, Yorkshire	1
Scarborough	1
Seaton Hall	1
Whitby	12
York	2
Yorkshire	4
Scotland	**16**
Dundee	2
Edinburgh	3
Glasgow	2
Kirkwall, Orkneys	1
Perth, Scotland	1
Scotland	7
Outside Britain	**4**
Cork, Ireland	1
Island of Minorca	1
Norway	2

Nativity	Individuals
Tyne and Wear	**47**
Boldon	1
Cleadon	1
Gateshead	8
Hetton-le-Hole	1
Houghton le Spring	5
Monkwearmouth	2
Newcastle	19
Sunderland	8
Wallsend	2
County Durham	**32**
Billingham	1
Bishop Aukland	3
Chester-le-Street	3
County Durham	4
Durham	2
Elwick	1
Ferryhill	1
Kibblesworth, Co. Durham	2
Lumley, Co. Durham	2
Middleton, Co. Durham	1
Raby, Co. Durham	1
Sedgefield	2
Shincliff, Co. Durham	1
Stockton-upon-Tees	5
Tanfield, Co. Durham	2
Whickham, Co. Durham	1
Wider Britain	**15**
Bordon, Hampshire	1
Cheshire	1
Clifton, Cumberland	2
Colchester, Essex	1
Ipswich, Kent	1
Leicestershire	1
Liverpool	1
London	5
Witwell, Derbyshire	1
Workington, Cumberland	1

Table 5.1: Location of birth for parents of children being baptised at St Hilda's, South Shields for the year 1798 (MF831).

The majority of migration was towards towns but there was the occasional instance of movement in the other direction. If the hypothesis were true, individuals moving from an urban area where congenital defects were more prevalent would increase the total number of defects that would otherwise have been seen in a rural population. Examples of migration to rural areas include an influx of migrant workers, particularly Irish individuals, to Yorkshire at harvest time (Pickles, 2002) and children taken from workhouses in nearby towns to supply a workforce for the newly opened textile mills in Fewston parish (Alred, 1997, 2001; Atkinson, 1989). Assuming the indigent nature of these children working in the mills, they would likely have been heavily exposed to a life with risks for health, such as poor maternal nutrition, infectious disease, and exposure to air pollution (see Section 2.3 and 5.2.2), all of which could have been teratogenic. These children, whether living to adulthood and settling in the area or passing away at a young age, could have theoretically contributed to a higher than expected congenital defect prevalence rate. Also affecting Fewston, "navvys," itinerant manual workers on civil engineering projects, moved into the parish for the building of the Fewston Reservoir (Harker, 1988; PR/FEW 1/19). As a moving workforce, there is no obvious way of knowing where these individuals were born but, according to parish records, some of the workers were buried in the churchyard of St Michael and St Lawrence (*e.g.* three men in 1871) (PR/FEW 1/19). Wharram Percy was not left out when it came to migratory workers. Around 200 miners and other railway workers descended on the parish from 1847 to 1853 for the construction of the Burdale railway tunnel (Bell and Beresford, 1987; Yorkshire Wolds Railway, 2013). Nearly half of the workers brought their families with them, as shown in the baptism records of the parish (Bell and Beresford, 1987; PR/WP 3).

As can be seen, the dynamic urban environment of North and South Shields attracted individuals from all over Britain and even other countries. While the majority of the populations at Fewston and Wharram Percy were likely to have been quite stable, in terms of migration there was the occasional influx of individuals from outside the community. If individuals born in urban locations do have a higher prevalence of congenital defects, individuals born in one context and

migrating to and being buried in another may have affected the data on congenital defects for each cemetery.

5.2.4 Socioeconomic Status

It may be possible to account for the frequency patterns seen in congenital defects through examination of socioeconomic status. In modern populations, individuals from lower socioeconomic groups are more likely to suffer from maternal ill-health (Agha *et al.*, 2013; Ebela *et al.*, 2011; McDade and Adair, 2001; Schell and Ulijaszek, 1999). Poorer individuals, today and in the past, are more likely to work in hazardous occupations, live in inadequate housing, and have poor nutrition (Acuña-González *et al.*, 2011; Agha *et al.*, 2013; Cannadine, 1993; Li *et al.*, 2013; Robinson, 1847; Schell and Ulijaszek, 1999), with aspects of all these conditions potentially teratogenic. In the 18th and 19th centuries, higher rates of women were from a low socioeconomic status than men and applied more commonly for poor relief. This was due to restrictions on the occupations in which women could work, thus limiting their ability to support themselves (Ellis, 2000; Johnson and Nicholas, 1997). As a study of congenital defects actually informs us about maternal health, a higher proportion of women in the lower socioeconomic groups, potentially more greatly affected by teratogens, could have a huge impact on the presence of congenital defects.

To investigate the possible effects of socioeconomic status on the individuals from the study sites, an attempt was made to assign each parish a social standing with the stipulation that individual and family conditions may be extremely variable within any socioeconomic group. Based on occupations recorded in the monthly meeting minutes (Table 2.3), the Quakers from North Shields consisted mainly of artisans and middle class individuals (MF176). The individuals from Wharram Percy were largely poor, the parish being one of the least prosperous areas of the East Riding, but some were well off enough to afford a burial inside of the church (Beresford and Hurst, 1990; Pickles, 1996; PR/WP 3). The fee for burial inside the church was one guinea, compared to 5 shillings for burial in the churchyard, roughly a quarter of the cost. Parish registers record labourers,

shepherds, farmers, and servants as the most common occupations (Table 2.5) (PR/WP 3). Unfortunately, assigning a socioeconomic status to the other parishes is more problematic. At South Shields the most commonly listed occupation was mariner (Table 2.3) (MF831). This is likely to represent working class individuals, but under there could be vast differences in living conditions between a ship-owner and a sailor, if both were described as a mariner. The ascription as made by a parish clerk or the mariner themselves might be extremely subjective and certainly not consistent. Beyond mariners, linking socioeconomic status to occupations becomes even less obvious. Occupations such as butcher, master mariner, mason, and painter could represent a range of socioeconomic groups depending on the context of each individual, generally representing a range from working to middle class. A similar situation occurs at Fewston. "Farmer" is the most commonly listed employment but this occupation can range from a tenanted farmer on rented land to a "gentleman" farmer owning large swathes of land (Table 2.4) (PR/FEW 1/16, 1/17). Much like the lesser occupations at South Shields, the occupations of people at Fewston such as stonemason, tailor, and blacksmith apply to people from the working class to middle class depending on each individual's situation. A tailor may own his shop and have several employees or a tailor may be the hired hand of such a shop. Analysing wills and probates or tax returns for each individual buried at each cemetery may make it possible to fully assign a socioeconomic status to some of the study sites, but that was beyond the scope of this thesis.

However, that is not to say socioeconomic status should be entirely ignored. Using what can be known from parish records, people buried at Wharram Percy can probably be assigned to the working class, North Shields to the middles classes, and South Shields and Fewston to the working to middle classes. Wharram Percy had the highest prevalence of combined congenital defects and the lowest socioeconomic class, which fits well with the theory that poorer individuals would have had more exposure to teratogenic agents. However, if this theory were to be followed correctly, North Shields would have had the lowest prevalence rate of congenital defects. Instead it was the second highest above South Shields and Fewston, which probably both had a lower socioeconomic status. For the

prevalence of combined congenital defects, there was no statistically significant difference between Wharram Percy and North Shields, the two sites that were more tightly assigned and furthest apart in terms of socioeconomic status. These results indicate that socioeconomic status of the parishes as a whole was unlikely to be the cause for the pattern of congenital defects observed. However, this conclusion may be altered, if the assumptions made here about the socioeconomic status of the study populations are proven incorrect through further research.

5.2.5 Aetiology of Congenital Defects

This study set the hypothesis that higher frequencies of congenital defects would be found in urban populations due to their greater exposure to detrimental living conditions. However, the use of congenital defects as an indicator of overall health in a population is problematic due to their aetiology. Congenital defects are caused by environmental or genetic factors or a combination of the two (Fan *et al.*, 2013; Schoenwolf *et al.*, 2009; WHO, 2012).

Looking firstly at environmental factors, just because a teratogen was present does not mean that it caused a congenital defect. The mother's exposure to the teratogen may not have occurred at the right time during pregnancy to cause a disruption, or the person may not have been exposed to the teratogen at a high enough dose or for long enough (Acuña-González *et al,.* 2011; Czeizel, 2008; Gluckman and Hanson, 2005; Holmes, 2011; Schoenwolf *et al.*, 2009). Additionally, in a location with multiple teratogens present, without the maternal history detailing any possible exposures, it is never possible to ascribe a specific teratogen to the presence of a congenital defect. Table 5.2 (an updated Table 2.1) shows teratogens known to affect the skeleton that may have been present in the past at the study sites. The anomalies highlighted in yellow are the congenital defects that were found in this study. The presence of these defects may indicate that a specific teratogen was present in these populations, but as multiple teratogens lead to the same defect, it is impossible to say that a specific teratogen was definitely present.

Category	Teratogen	Defects
Maternal Conditions	Diabetes	neural tube defects, absent lumbar or sacral elements, femoral head aplasia, heart defects, brain defects
	Hypervitaminosis A	craniosynostosis, calvarium hypoplasia, mandible malformations, cleft palate, craniofacial defects, supernumerary vertebrae, absent vertebrae, premature long bone fusion, cardiovascular defects, central nervous system defects
	Zinc Deficiency	cleft lip and palate, anencephaly, neural tube defects, spina bifida, absent vertebrae, fused thoracic and lumbar vertebrae, spinal curvatures, rudimentary ribs, fused ribs, syndactyly, clubbed feet, agenesis of limbs
	Iron	spine defects, rib defects
	Manganese	domed skull, shortened long bones, dysplasia of tibial epiphyses
Infectious Disease	High Fevers	cleft lip and palate, microphthalmia, facial defects, microcephaly, neural tube defects, neurogenic limb contractures, external genital defects, external ear defects
	Rubella	microcephaly, heart defects, mental handicap
	Lymphocytic Choriomeningitis Virus	encephalitis, microcephaly, macrocephaly, hydrocephalus, neurological defects
Heavy Metals	Selenium	spinal curvatures
	Stannous Chloride	microcephaly, microphthalmia
	Benzo(a)pyrene	craniofacial defects
	Lead	fused cervical vertebrae, delayed ossification
	Arsenic	cleft lip and palate, neural tube defects, fused vertebrae, occipitalization, fused ribs
	Mercury	cleft lip and palate, encephaly, fused ribs, clubbed feet, syndactyly
Other	Hypoxia	cleft lip, craniofacial defects, neural tube defects, limb reduction defects, heart defects, brain defects
	Carbon Monoxide	microcephaly, skull defects, mandible defects, wedged vertebrae, fused vertebrae, absent vertebrae, hemivertebrae, scoliosis, fused ribs, lumbar ribs, brain damage
	Carbon Dioxide	vertebral defects, ectrodactyly, heart defects

Table 5.2: Summary of teratogens and the congenital defects they may cause. Defects in blue may affect the skeleton and be seen archaeologically. Defects in yellow were found at the study sites.

Even if specific teratogens could be linked to particular defects, it is the fact that genetic causation can never be dismissed which means that congenital defects

on their own are not likely to be convincing indicators of overall levels of health or disease. The defects that are passed on through a genetic mutation can also never be excluded from those caused by maternal exposure to teratogens. The genetic factor is perhaps what has led to the wider range of prevalence rates for congenital defects at the study sites. Skeletally, humans generally react to infection, parasite infestation, and nutritional "stress" in much the same way. In addition to actually causing defects in and of themselves, a genetic predisposition can also make a fœtus more or less susceptible to teratogens *in utero* (Holmes, 2011). Two women in the same stages of pregnancy exposed to the same level of a teratogen can have two very different outcomes to their pregnancy. One infant may be born healthy with no defects, while the other may be born with very obvious signs of exposure to a given teratogen.

5.2.6 Congenital Defects Conclusions

This section has attempted to meet three of the aims and objectives of this thesis, to assess the impact of teratogenic agents on 18th and 19th century populations in Northeast England, to assess the importance of settlement type on frequency of congenital defects, and to provide a better understanding of the effects of health and living environment on the occurrence of congenital defects. As has been shown, the pattern seen for the presence of congenital defects at the four study sites is not easy to explain. The lack of an urban/rural divide can be partly attributed to similar poor living conditions occurring at both urban and rural sites in the 18th and 19th centuries even if the high population densities of urban areas may have exacerbated the health risks. However, migration of individuals between contexts may slightly bias the findings, with urban individuals settling in rural areas raising the prevalence rates and with the opposite effect occurring in urban areas. Socioeconomic standing of a parish, meanwhile, seems to have had little effect on the occurrence of congenital defects at the study sites. When undertaking studies such as this is it important to remember that genetics plays a role in the development of congenital defects so that on their own, these anomalies may not serve as good indicators of health.

5.3 "Stress" Indicators

The presence of "stress" indicators such as porotic hyperostosis (PH), cribra orbitalia (CO), dental enamel hypoplasia (DEH), and periosteal new bone formation (PNBF) along with stature can help to assess an individual's or population's exposure to poor nutrition and chronic or repeated bouts of illness (Goodman *et al.*, 1988; Lewis and Roberts, 1997; Obertová and Thurzo, 2008; Šlaus, 2000; Sullivan, 2005). These indicators were used to determine whether poor living conditions seen in the written documentation for the study sites (Section 2.3) could also be seen in the skeletal populations. Differences in the presence of these "stress" indicators may indicate variations in living conditions occurring at the four study sites. Below, the findings for "stress" indicators and stature are reviewed, followed by a discussion on the possible meanings of these patterns.

5.3.1 Results Summary

There were no statistically significant differences found between the four sites, or between combined urban and rural sites, for the presence of all "stress" indicators combined. In fact, all sites had extremely similar prevalence rates. Wharram Percy had the highest (56.90%), followed by North Shields (56.30%), South Shields (52.85%), and Fewston (52.55%). Similarly, for PNBF of the tibia and fibula, DEH, and PH there were no significant differences between rural and urban sites. Nearly statistically significant results (significant at 90%) were found when comparing tibial periosteal new bone formation in individuals from Fewston and those from the combined urban sites. While the Fewston population had a lower frequency of tibial periosteal new bone formation, individuals from South Shields had a higher frequency of tibial periosteal new bone formation than those from the combined rural sites. However, these data were not seen for PNBF of the fibula.

The data for CO produced the only statistically significant results. Individuals from Fewston had the highest prevalence of CO when compared to all other individual sites and combined urban sites when looking at both the number of individuals affected and number of orbits affected (although this was not significant when compared to Wharram Percy individuals) (Table 5.3). Both individual urban

sites had statistically significant differences when compared to the frequencies of CO at the combined rural sites. In recording CO, the appearance (healed, in a state of healing, or still active) of the lesions was not reported as this study did not require this specific information. As CO was used as one of several indicators of overall health in the population, for the purposes of this study it did not matter at what stage in life someone was affected by CO. The important point to make is that it occurred at all, therefore demonstrating that the stressors required to produce this change were present at that settlement site during that person's lifetime.

Site		vs WP	vs NS	vs SS	vs Rural	vs Urban
Fewston	Individ	0.1006	0.0062	0.0004	-	0.0006
	Orbit	0.0228	0.0002	0.0001	-	0.0001
Wharram Percy	Individ	-	0.8330	0.3370	-	0.5248
	Orbit	-	0.7359	0.2700	-	0.5022
North Shields	Individ	-	-	0.2410	0.0372	-
	Orbit	-	-	0.3455	0.0026	-
South Shields	Individ	-	-	-	0.0021	-
	Orbit	-	-	-	0.0002	-
Rural	Individ	-	-	-	-	0.0032
	Orbit	-	-	-	-	0.0002

Table 5.3: Statistical analysis (two-tailed p values) of the presence of cribra orbitalia as recorded both by individual and by orbit. Yellow indicates a statistically significant result ($p \leq 0.05$).

Attained adult stature is determined by genetics but can be limited or enhanced by environmental factors (Arcini *et al.*, 2012; Bielicki, 1998; Briend, 1998; Haviland, 1967; Schell, 1998; Steckel and Floud, 1997; Watts, 2011). Poor nutrition, ill health, and environmental pollution can have an effect on the final height an individual reaches. When comparing stature between individuals from the combined urban and rural sites, there was no statistically significant difference. Males from Fewston were statistically taller than males from any other individual site, but the females were the shortest (not statistically significant). No statistically significant results were found between sites for the females. As has been seen in the discussion of congenital defects and other "stress" indicators, malnutrition, infectious disease, and air pollution were rife in both the urban and rural sites during the study period. All of these conditions could have been detrimental to the

growth of individuals living in these locations leading to a shorter than genetically expected stature.

It should be pointed out that there may be some skewing of results due to the method involved in determining stature. Each long bone used to estimate stature has a different error range (Trotter, 1970). As detailed in Section 3.4.4, long bones used for measurement with the lowest error values were preferentially chosen. However, due to preservation issues, the same bone could not be used for every individual, leading to bones with higher error values being used for certain measurements. Error values for each individual's stature are provided in Appendices B through E.

5.3.2 Congenital Defect Comparison

When looking at combined "stress" indicators and combined congenital defects, the frequency patterns for the four sites are similar for both (Figure 5.1). The ranges of prevalence rates are different, with the difference between sites much less for "stress" indicators (52.55-56.90%) than that seen for congenital defects (18.54-43.33%). This further indicates that congenital defects could potentially serve as a "stress" indicator. However, as discussed in Section 5.2.5, this is perhaps not a realistic possibility due to the genetic component of congenital defect causation.

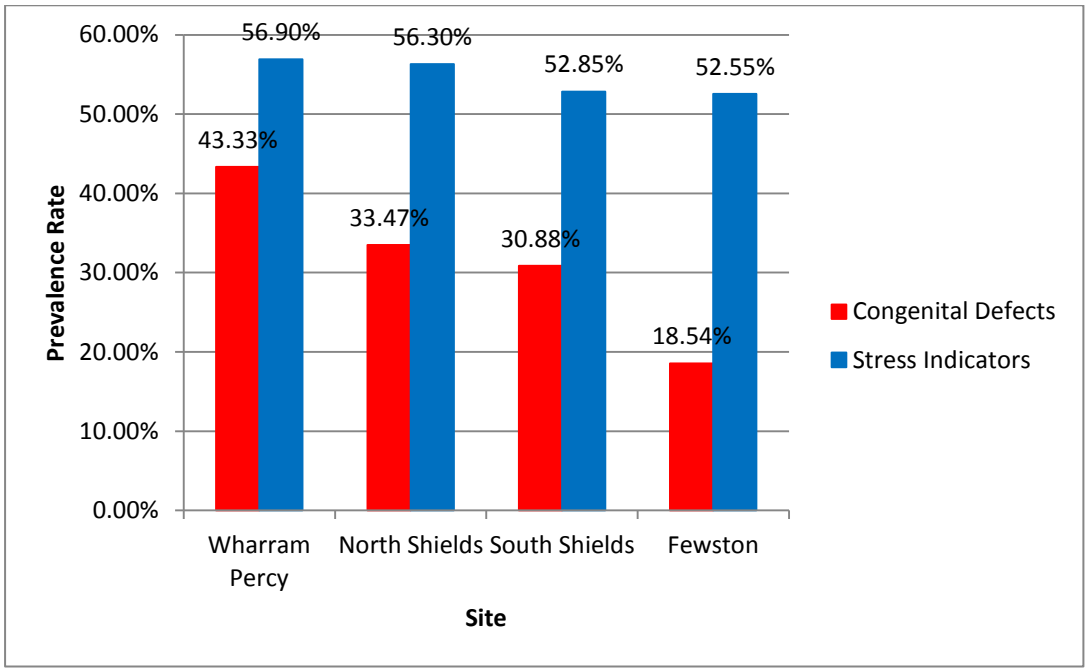

Figure 5.1: Prevalence rates for combined congenital defects and combined "stress" indicators.

Looking at the frequency rates for "stress" indicators and congenital defects for individual sites, Fewston stands out. Fewston had the lowest prevalence rate of combined congenital defects (and commonly the lowest prevalence rates for individual defects as well) and combined "stress" indicators. Additionally, the males from Fewston were the tallest in the study (statistically significant). These all point to individuals living in the healthiest environments out of the four study sites. However, Fewston also had the highest prevalence of CO (statistically significant) and the shortest females, although the latter finding was not statistically significant. Contradicting the data above, both of these findings would indicate Fewston parish was the least healthy environment. As stature is ultimately controlled by genetics, the females from Fewston may have been healthy and well-nourished but their growth would never have reached that seen at the other locations if Fewston had the genetically shortest females. Alternatively, there may be a link with CO. Comparing CO in females and males at Fewston for individuals that could be sexed and examined for CO, females (11/51, TPR=21.57%) were most affected compared to males (4/51, TPR=7.84%). This difference was not quite statistically significant ($p=0.0911$, significant at 90%). This suggests there may have been a higher incidence of CO amongst the female population of Fewston. The malnutrition or

disease that caused the CO may have stunted the growth of the females in an otherwise healthy environment.

5.3.3 Living Conditions

The most likely cause for the lack of a statistical significance for prevalence rates between urban and rural sites for "stress" indicators is that poor living conditions were actually very similar between the two types of site. This was explored with reference to congenital defects above in Section 5.2.2. Since all four sites contained a similar prevalence rate for "stress" indicators, it is reasonable to suggest that all four locations had similar levels of "stress". PH and CO are an expansion of the diploë of the cranium in response to anæmia potentially caused by a number of aetiological factors such as parasitic infection, blood loss, malabsorption of iron, and diarrhoeal diseases (Grauer, 1993; Martin *et al.*, 1985; Obertová and Thurzo, 2008; Šlaus, 2008; Stuart-Macadam, 1992a, b; Sullivan, 2005; Walker *et al.*, 2009; Wapler *et al.*, 2004). Parasitic infections and diarrhoeal diseases would have been endemic in the unsanitary, overcrowded conditions of urban areas, and the unsanitary conditions and association with livestock in rural areas (Addyman, 1989; Alred, 1997; Harker, 1988; Hey, 1986; Hodgson, 1903; Hood Coulthard, 1959, 1960; Jennings, 1967; Reid, 1845a, b; Simpson, 1988; Southern, 2003).

DEH is an enamel defect caused by an arrest in development of tooth formation due to a prolonged period of disease or malnutrition in early childhood (Aufderheide and Rodriguéz-Martín, 1998; Garcin, *et al.* 2010; Goodman, 1998; King *et al.*, 2005; Liebe-Harkort, 2012; Rose *et al.*, 1985). Infectious disease was prevalent in both urban and rural locations in the post-medieval period due to close proximity to other humans and/or livestock (Harker, 1988; Hey, 1986; Hodgson, 1903; Horsley, 1971; Openshaw, 1978; Wrightson, 2009). Malnutrition would have been very likely on the limited diet available to many of the working class individuals (Drummond and Wilbraham, 1957; Hey, 1986; Ketabgian, 2007; Wilson, 2003). Furthermore, beliefs in the safety of some foods led to restricted diets even in those further up the socioeconomic ladder (Drummond and Wilbraham, 1957;

Freeman, 1989; Giorgi, 1997; Thirsk, 2006; Wilson, 2003). PNBF of the tibia and fibula may be caused by infection, ulceration, or trauma (Assis *et al.*, 2011; Boel and Ortner, 2013; Grauer, 1993; Šlaus, 2008; Weston, 2008, 2012). Again, infection and infectious disease would have been common in both urban and rural environments during this time period.

5.4 Site Comparisons

In order to assess how typical the results found in this study are, they need to be compared to other contemporaneous populations. Urban sites were compared to combined urban and rural prevalence rates to look for similarities or differences in an effort to better explain the patterns observed in this study. Unfortunately, no suitable rural sites were available for comparison. The sites of North Shields and Wharram Percy allowed for special comparisons. Individuals from North Shields were compared to other Quaker burial grounds, and the post-medieval individuals from Wharram Percy were compared to their medieval counterparts buried in the same cemetery.

5.4.1 Comparisons of Urban Environments

A. Birmingham

A good comparison for a contemporaneous cemetery population from an industrial northern town is St Martin's-in-the-Bull-Ring, Birmingham. A skeletal sample of 505 individuals dating from the late 18th to early 19th centuries was examined (Brickley *et al.*, 2006a). The individuals were largely working class with some coming from the middle class. Living conditions at the time these individuals were alive, were generally better than most major towns of the time, but the worst conditions were found in the older, densely-packed areas of town, such as those found around St Martin's (Brickley *et al.*, 2006a). These older courts tended to be badly drained, ill-ventilated, and filthy.

Congenital defects recorded for individuals from St Martin's, Birmingham were hemimetameres, cleft lip/palate, cervical ribs, cleft neural arches (thoracic, lumbar, and sacral), supernumerary vertebrae (T13 and L6), occipitalization, Klippel-Feil syndrome, sacralization, lumbarization (S1 and T12), scoliosis, and assorted rib anomalies. The overall number of individuals affected by at least one defect is not reported but excellent data are provided for nearly all defects recorded allowing for comparisons by defect. Comparing the current study's data from combined rural and urban sites with Birmingham's data, there was no significant statistical difference in the occurrence of cervical ribs, occipitalization, lumbarization of S1, thirteen thoracic vertebrae, cleft neural arches (thoracic), six lumbar vertebrae, Klippel-Feil syndrome of the cervical vertebrae, and rib anomalies. Clefting of the lumbar vertebrae was more common in people buried in the urban sites in this study (12 out of 1148, 1.05%) compared to Birmingham (1 out of 1479, 0.06%). This difference was extremely statistically significant (p=0.0004). There was no difference when comparing Birmingham with the rural sites in this study (1/394, 0.25%). Two observed border shifts are of particular note: lumbarization of T12 and sacralization of L5 or L6. For both shifts, the individuals from combined urban and rural sites had higher prevalence rates than at Birmingham. For lumbarization of T12, the urban (59/241, 24.48%, p=0.0001) and rural sites in this study (15/83, 18.07%, p=0.0001) had more occurrences than at Birmingham (4/294, 1.36%), which was found to be extremely statistically significant. Results were similar with sacralization, where urban (34/257, 13.23%, p=0.0247) and rural frequency rates (14/83, 16.87%, p=0.0177) were higher than those at Birmingham (23/311, 7.40%) (statistically significant).

Given the possibility of similar socioeconomic backgrounds of the individuals buried at Birmingham and South Shields (see Section 5.2.4), it may be worth comparing the two sites individually. For most defects, there were no statistically different results. For four defects (L6, cleft neural arch of the lumbar vertebra, lumbarization of T12, and sacralization of L5 or L6), the data showed a statistically significantly higher prevalence rate at South Shields (Table 5.4). These data largely mirror those found for the combined urban sites.

Defect	Site	Affected	Observable	Prevalence	Statistics Result
L6	Birmingham	17	303	5.61%	p=0.0138
	South Shields	16	118	13.56%	
Cleft Neural Arch - Lumbar	Birmingham	1	1479	0.06%	p=0.0011
	South Shields	6	477	1.26%	
Lumbarization of T12	Birmingham	4	294	1.36%	p=0.0001
	South Shields	25	97	25.77%	
Sacralization of L5 or L6	Birmingham	23	311	7.40%	p=0.0345
	South Shields	16	110	22.72%	

Table 5.4: Individuals affected, prevalence rates, and statistical results for the comparison between Birmingham and South Shields. Data for Birmingham from Brickley *et al.* (2006a). Yellow denotes a statistically significant result (p≤0.05).

The "stress" indicator cribra orbitalia was also recorded at St Martin's, Birmingham. When comparing cribra orbitalia from the individuals observable for the trait at Birmingham (38/394, 9.64%), individuals from the urban (57/343, 16.62%, p=0.0057) and rural sites (35/118, 29.66%, p=0.0001) in this study both proved to have statistically higher rates.

B. Barton-upon-Humber

A second comparison that can be made between this study and contemporaneous populations comes from north Lincolnshire. The cemetery of St Peter's, Barton-upon-Humber was in use from the late 10th century (Waldron and Rodwell, 2007). Due to its relative geographical isolation, the town never transformed into a major regional centre and never experienced rapid growth until the early 19th century. Small scale commerce was Barton-upon-Humber's major industry with brick and tile-making starting in the late 17th century. The population in the 17th century was a little over 1,000, only rising to 1,709 in 1801 (Waldron and Rodwell, 2007).

For the purposes of this comparison, only the post-medieval individuals were examined. Of the excavated individuals, 729 were dated to about 1500 to 1855 AD. Congenital defects recorded in the population included os acromiale, spondylolysis, congenital dislocation of the hip, spina bifida occulta (here meaning clefting of the entire sacrum), transitional vertebrae at the lumbosacral border, microtia (aplasia of the auditory meatus), idiopathic scoliosis, and Klippel-Feil

syndrome. Due to the information provided in the skeletal report, only crude prevalence rates for os acromiale, spondylolysis, scoliosis, and "spina bifida occulta" could be compared to data from this study. For these three defects, no statistical significance could be found in the crude prevalence rates for the individuals from the combined rural and urban sites in this study and those from Barton-upon-Humber.

C. Cross Bones, London

At the Cross Bones burial ground, Southwark, London, 148 individuals were excavated dating to the middle of the 19th century (Brickley *et al.*, 1999). At that time, the burial ground was used to inter the parish poor. The parish consisted of densely-packed and poorly-built houses lined along filthy streets (*ibid.*). Families typically shared one room, while drainage systems either did not exist or were badly maintained leading to accumulation of filth. Water for this area was obtained directly from the River Thames, nutrition was poor, and fevers were endemic to the area.

Congenital defects recorded for the individuals buried at Cross Bones were spina bifida occulta (here meaning clefting between halfway down the sacrum and the fifth segment), sacralization, lumbarization (both T12 and S1), os acromiale, thirteen thoracic vertebrae, six lumbar vertebrae, scoliosis, anterior clefting of the first cervical vertebra, Klippel-Feil syndrome, rudimentary transverse process, aplastic transverse process, lumbar rib, and bifid ribs (Brickley *et al.*, 1999; WORD Database, 2013b). A mix of crude and true prevalence rates was available for comparison for several of the defects. No statistical difference was found between Cross Bones and this study for the presence of os acromiale (comparing true prevalence rates) or lumbar ribs (comparing crude prevalence rates). The statistically significant results can be found in Table 5.5. All statistically significant or nearly statistically significant results, except those for bifid ribs, show a higher defect rate at urban or rural sites in this study. For the presence of a bifid rib, Cross Bones individuals had statistically significant higher crude prevalence rates.

Defect	Site	Affected	Individuals	CPR	Statistics Result
Lumbarization of T12	Cross Bones	8	148	5.41%	-
	Urban	59	449	13.14%	p=0.0101
	Rural	15	211	7.11%	p=0.6625
Sacralization	Cross Bones	3	148	2.03%	-
	Urban	34	449	7.57%	p=0.0167
	Rural	14	211	6.64%	p=0.0463
L6	Cross Bones	2	148	1.35%	-
	Urban	26	449	5.79%	p=0.0246
	Rural	12	211	5.69%	p=0.0502
Bifid Rib	Cross Bones	4	148	2.70%	-
	Urban	2	449	0.45%	p=0.0357
	Rural	0	211	0%	p=0.0282

Table 5.5: Individuals affected, prevalence rates, and statistical results for the comparison between Cross Bones and the urban and rural sites of this study. Data for Cross Bones comes from WORD Database (2013b). Yellow denotes a statistically significant result ($p \leq 0.05$) and green denotes a nearly statistically significant result ($p \leq 0.10$).

The only "stress" indicator recorded at Cross Bones that was comparable with the data in this study was cribra orbitalia. Individuals from Cross Bones showed statistically significantly higher true prevalence rate for the presence of cribra orbitalia than the urban (57/343, 16.62%, p=0.0001) or rural individuals (35/118, 29.66%, p=0.0138) in this study.

D. Chelsea Old Church, London

Chelsea Old Church, London was excavated in 2000. A total of 290 skeletons were exhumed but only 198 of those were osteologically examined (WORD Database, 2013a). The cemetery contained mostly high status individuals who lived on the outskirts and more rural areas of London. Congenital defects recorded at Chelsea Old Church were os acromiale, sacralization, lumbarization of both T12 and S1, the presence of a sixth lumbar vertebra, cleft neural arches of the sacrum, scoliosis, spondylolysis, rudimentary apophyseal facet, and bifid ribs. For the defects os acromiale, lumbarization of S1, presence of a sixth lumbar vertebra, cleft neural arches of the sacrum, spondylolysis, and bifid ribs, there were no statistically significantly different results when compared to the combined sites in this study. Statistically significant results were found for sacralization, lumbarization of T12, and scoliosis (Table 5.6). Individuals from Chelsea Old Church had a statistically

higher crude prevalence rate of scoliosis compared to both the urban and rural sites from this study. For the other two defects, the frequency rates in this study proved to be higher.

Defect	Site	Affected	Individuals	CPR	Statistics Result
Sacralization	Chelsea	5	198	2.53%	-
	Urban	34	449	7.57%	p=0.0117
	Rural	14	211	6.64%	p=0.0600
Lumbarization of T12	Chelsea	1	198	0.51%	-
	Urban	59	449	13.14%	p=0.0001
	Rural	15	211	7.11%	p=0.0005
Scoliosis	Chelsea	7	198	3.54%	-
	Urban	3	449	0.67%	p=0.0113
	Rural	1	211	0.47%	p=0.0323

Table 5.6: Individuals affected, prevalence rates, and statistical results for the comparison between Chelsea Old Church and this thesis's urban and rural sites. Data for Chelsea Old Church from WORD Database 2013a. Yellow denotes a statistically significant result (p≤0.05) and green denotes a nearly statistically significant result (p≤0.10).

E. St George's Church, Bloomsbury, London

The seven vaults located in the crypt of St George's Church, Bloomsbury contained 781 burials dating from 1804 to 1856 (Boston *et al.*, 2009). One hundred and eleven of these individuals were found in open lead coffins and analysed on site. Of these individuals, 72 were named and 39 were not. This crypt contained individuals from the wealthy middle class but also tradesmen. Looking at just the 72 named individuals, spina bifida occulta (here defined as a completely clefted sacrum), cleft neural arch, sacralization, congenital scoliosis, block vertebrae, congenital hip dislocation, and fusion of the cuneiforms were the congenital defects reported. Statistical comparisons could be made between this study and that for St George's Church for a completely clefted sacrum, cleft neural arch, sacralization, and scoliosis. Only the clefted sacrum returned statistically different results, showing a higher crude prevalence rate of the defect at St George's (5/72, 6.94%) from the urban (1/449, 0.22%, p=0.0002) or rural sites (0/211, 0%, p=0.0010) in this study.

F. Stature Comparisons

Stature data were available from five post-medieval sites for comparison. St Martin's, Birmingham recorded means of 171.8cm for males and 159.1cm for females (Brickley *et al.*, 2006b). St Peter's, Wolverhampton reported means of 171.0cm for males and 160.6cm for females (Adams and Colls, 2007). At Chelsea Old Church, London, means of 168.4cm for males and 163.4cm for females were observed (WORD Database, 2013a). Also in London, St Benet Sherehog reported means of 169.4cm for males and 160.2cm for females (Miles *et al.*, 2008b). Individuals from the Quaker burial ground, King's Lynn returned means of 168.7cm for males and 160.3cm for females (Bashford and Sibun, 2007). These five combined sites give a range of 168.4-171.8cm for males and 159.1-163.4cm for females. The statures recorded in this study all fit within this range with the exception of individuals from Fewston. The males from Fewston were taller with a mean of 176.43cm and the females were shorter with a mean of 158.99cm.

G. Overview of Urban Comparisons

Comparisons of congenital defects and "stress" indicators here show that the urban populations of this study were largely similar to those of other towns of the period across Britain. Unfortunately, it also shows that the rural sites were also similar to these urban areas. This exercise suggests that the experiences of people living across England from different socioeconomic groups during the post-medieval period were similar to each other. Many towns of the period had shared industrial practices producing similar levels of smoke and other pollutants. In the urban areas, the large scale industries quickly brought together great numbers of people to settle in a small area, leading to crowded housing, poor sanitation, and endemic infectious diseases. When looking at the urban/rural divide, the differences been urban and rural areas for this time period may not be as marked as one would have thought given the level of detailed discussion in historical literature on the harmful living conditions encountered in urban areas in the industrial period (Ellis, 2001a; Reid, 1845a, b; Robinson, 1847; Sharpe, 2000). This may, however, be a modern reaction to a historical bias in the reporting of conditions in the industrialised urban

areas due to a political and moral urgency to reform those conditions (see Section 5.6.5 below).

5.4.2 Comparison of Quaker Burial Grounds

A. Kingston-upon-Thames

In 1996, a Quaker burial ground was excavated in Kingston-upon-Thames, Greater London (Bashford and Sibun, 2007; Start and Kirk, 1998). Excavations uncovered 360 individuals buried between 1664 and 1814, a time contemporaneous with the North Shields population. The Kingston-upon-Thames meeting represented a mercantile middle class in an urban settlement. Four types of congenital defects were reported in the population, one undiagnosed disorder involving shortening of the humeri and an abnormally broad skull, and three affecting the spine. Four individuals were found to have supernumerary vertebrae (three with a T13 and one with a C8). At North Shields, no individuals were recorded with a T13 or C8 but ten individuals had an additional lumbar vertebra, while three had an additional sacral element. Comparing crude prevalence rates between the two populations shows a very statistically significantly higher rate of supernumerary vertebrae at North Shields ($p=0.0042$). Two individuals at Kingston had sacralization of the L5 while nine at North Shields showed sacralization of either the L5 or L6. Again, the crude prevalence rates are very statistically significant, with a higher rate at North Shields ($p=0.0092$). The final vertebral defect observed at Kingston was scoliosis in one female, whereas North Shields had two individuals with scoliotic changes. Statistically, there is no difference between the sites for this defect. For all congenital defects, North Shields (82/245, 33.47%) proved to have a higher rate when compared to Kingston (12/360, 3.33%) ($p=0.0001$, extremely statistically significant).

While not perfect data, being largely based on crude prevalence rates, the trend seems to show a higher rate of congenital defects at North Shields than at Kingston-upon-Thames. This may be due to differential living conditions. While Kingston is on the fringes of London, its location to the west, in the opposite

direction of prevailing winds, likely shielded it from the brunt of the pollution and overcrowding occurring in the city centre and other heavy industrial areas (Bashford and Sibun, 2007). Individuals living in North Shields would not have been so fortunate. As an industrial centre, the population would not have been spared the harmful effects of air/water pollution, poor sanitation, dietary deficiencies, and infectious disease. These different living conditions can be seen in the skeletons themselves. At Kingston, no evidence of dietary deficiencies or poor living conditions was reported within the skeletal population. In contrast, 56.30% of the individuals buried at North Shields had at least one indicator of "stress". For the Kingston data, all burials were examined for age and sex, and gross pathological information, but only 36% of the burials were fully recorded. It is possible to assume some occurrences of "stress" indicators may have been missed due to the inadequate recording process, but it seems unlikely that they would have been missed for every skeleton of the 137 or so individuals fully recorded. Alternatively, the population may have been too frail to withstand the environmental stresses they were exposed to, leading to death before an osteological response could have occurred (see Section 5.6.2 below; Wood *et al.*, 1992). But again, it seems unlikely that the entire population would have been that frail. Therefore, it may be said the individuals from North Shields and Kingston-upon-Thames can be seen as representing two areas with very different living conditions. This can be seen skeletally in the differences in prevalence rates for congenital defects and "stress" indicators.

B. King's Lynn

The Friends burial ground at King's Lynn is an interesting cemetery for comparison. No dates for use of the cemetery are given but it appears on the Ordnance Survey map in 1830 (Mahoney, 2005). Of the 34 middle-class and largely mercantile individuals excavated from the burial ground, five showed evidence of a congenital defect (14.71%). Defects that were found included a congenitally fused rib, a cervical rib, hypoplastic foot bones, an unusual auricular surface, and an individual with slight scoliosis. Individuals from North Shields appeared to be much

more greatly affected by congenital defects (33.47%), and this difference was statistically significant (p=0.0294).

Unlike at Kingston-upon-Thames, "stress" indicators were recorded although they were not high in number. PNBF of the tibia and fibula, CO, and DEH were all seen at King's Lynn and one or more of these conditions were recorded in 11 individuals (32.35%). This prevalence was much lower than that seen for North Shields (56.30%), and the difference proved to be statistically significant (p=0.0101). Given the limited number of individuals with "stress" indicators and the pathologies present at King's Lynn, the researchers report no evidence for dietary deficiencies and sparse evidence for poor living conditions.

King's Lynn is in a similar geographical situation to that of North Shields in that it is located on the River Great Ouse near where it joins the North Sea. As such, much like North Shields, the dominant industry was shipping and its associated industries such as rope and sail making (Hodgson, 1903; Hood Coulthard, 1960; Mahoney, 2005; Phillips, 1894; Sykes, 1866a). One major difference however, was the lack of coal production in and around King's Lynn. This would have led to a dearth of the heavy coal smoke producing industries such as salt and glass making seen in North Shields.

C. St Ives

Excavations in 2006 uncovered sixteen individuals from a Quaker burial ground in St Ives, Cambridgeshire located along the River Great Ouse (Clough and Loe, 2007). The burial ground was in use from approximately 1687 to 1721, so mostly predating that of North Shields. Congenital defects recorded for the population included sacralization, supernumerary vertebrae (L6 and S6), "spina bifida occulta" (actually cleft neural arches), aplasia of several elements of vertebrae, scoliosis, and a cervical rib. Five individuals were affected by congenital defects at St Ives (31.25%). This is a similar prevalence rate, just slightly lower, to that found at North Shields (33.47%) and, unsurprisingly, the marginal difference is not statistically significant (p=1.0000). Six individuals at St Ives displayed the

"stress" indicators CO, DEH, and/or tibial PNBF (37.50%) which is much lower than the rates found at North Shields (56.30%), but these differences were not statistically significant (p=0.1944).

While the numbers are very small, it is worth mentioning that two sixth lumbar vertebrae were present and of which one was sacralized with a cleft. At North Shields the majority of L6 were sacralized and half of those sacralized were clefted. In fact, the majority of sixth lumbar vertebrae recorded in this study from all four sites were sacralized with at least a portion of them also exhibiting clefting, but North Shields had the highest percentage. Seeing this trend at multiple sites suggests a further study of the relationship between L6, sacralization, and cleft neural arches could prove enlightening.

No information was provided on living conditions of the St Ives population, or the perceived socioeconomic status of the individuals interred in this Quaker burial ground in the skeletal report. Using the skeletal data, it can be seen that individuals from North Shields were perhaps slightly more affected by their poorer living conditions. As these results are not statistically significant, this is not conclusive and the lack of other types of evidence makes it difficult to compare the lives of individuals from St Ives to those of individuals from North Shields.

D. Overview of Quaker Sites

Comparing individuals buried at North Shields, Kingston-upon-Thames, and King's Lynn provides an interesting picture. (*N.B.* As no information on living conditions or socioeconomic status was given for St Ives, this site will not be included in this discussion.) Given the similar levels of health at Kingston, the researchers for King's Lynn suggest 'that the good health and low-"stress" lifestyle of this population is characteristic of Quaker communities and not necessarily that of the surrounding population' (Mahoney, 2005:108). However, as shown above, individuals from North Shields have higher rates of congenital defects than those at either Kingston or King's Lynn, although not all of these data are statistically significant. The prevalence rates recorded for North Shields were also not that

dissimilar to those found amongst the Anglican population buried at South Shields. North Shields Quakers had more in common health-wise with their immediate neighbours across the river than with those from their religious community elsewhere in the country. Given the similar religious beliefs and socioeconomic standing (largely mercantile middle class) between the three Quaker communities, the answer must lie in living conditions and environment. Both Kingston and King's Lynn appear to have escaped much of the pollution and other poor living conditions reported at North Shields during this time. The ever-present coal smoke found permeating North Shields would have affected everyone in the area, regardless of social standing. In short, religion and even socioeconomic standing were not enough to protect Quakers in North Shields from detrimental living conditions and health consequences.

5.4.3 Wharram Percy: Congenital Defect Frequency Through Time

Using previously recorded data, it is possible to look at the prevalence of congenital defects through time at Wharram Percy. Sture (2001) observed congenital defects and "stress" indicators in 377 individuals dating from the 13th to 16th centuries. Congenital defects found at Wharram Percy in the late medieval period can be compared to those seen in the post-medieval period in Table 5.7. In total, 102 individuals buried in the late medieval period were found to have at least one congenital defect. This led to a prevalence rate of 27.06% compared to the 43.33% rate found for the post-medieval period. This increase in prevalence of congenital defects through time is statistically significant (p=0.0141). However, it is perhaps not a fair representation. As seen in Table 5.7, individuals buried in the post-medieval period were examined for defects of the spine and thoracic cavity. When directly comparing like for like, the statistical significance vanishes. The number of individuals affected by at least one congenital defect in the post-medieval period falls from 26 (43.33%) to 22 (36.67%). When comparing this new figure to the late medieval data, the p value becomes 0.1265 which is not statistically significant.

Late Medieval (Sture 2001)		Post-Medieval	
Spine:	**Thoracic Cavity:**	**Spine:**	**Thoracic Cavity:**
-lumbarization	-lumbar ribs*	-lumbarization	-lumbar ribs
-sacralization	-cervical ribs*	-sacralization	-cervical ribs
-spina bifida occulta		-spina bifida occulta*	-rib spur
-cleft neural arch		-cleft neural arch	-rudimentary rib
-block vertebra		-block vertebra	
-supernumerary vertebra		-supernumerary vertebra	**Appendicular:**
-spondylolysis		-spondylolysis*	-os acromiale
-absent vertebra		-absent vertebra	-aplasia ulnar styloid process
-spondylolythesis		-rudimentary transverse process	
		-rudimentary costal facet	

Table 5.7: Congenital defects observed at Wharram Percy in the late and post-medieval periods. *denotes a defect examined for but not seen in the population.

For Sture's (2001) research, the late medieval population from Wharram Percy was compared to other contemporary sites in and around York. For combined congenital defects, Wharram Percy individuals were similar to those from Raunds Furnells, Northamptonshire, the other rural site which was earlier in date (mid-10th to late 12th centuries) to Wharram Percy, and were different from two of the three urban sites, but no figures or statistics were given to support this observed trend (*ibid.*). The urban sites of the hospital of St James and St Mary Magdalene, Chichester, West Sussex and St Helen-on-the-Walls, York both had provided higher numbers of congenital defects than rural Wharram Percy (*ibid.*). When looking at "stress" indicators, Wharram Percy had the lowest rates for "stress" indicators (cribra orbitalia, dental enamel hypoplasia, and tibial PNBF) compared to contemporary sites although, again, no hard numbers or percentages were given in the report. Both of these trends are reversed in the post-medieval population, where Wharram Percy showed the highest rates of both congenital defects and "stress" indicators compared to the two urban and one rural sites.

A possible explanation for these observed changes is that the living conditions in the parish of Wharram Percy declined over time. The rise of industry in Yorkshire extended into all regions, affecting the lifestyle of inhabitants in urban and rural settings alike. Alternatively, the living conditions in the towns of the

Northeast may have improved over time. Knowledge of the harmful effects of living at a high population density and improper sanitation led to improvements in the overall health of the urban populous (Hodgson, 1903; Linsley, 1992; Reid, 1845a, b; Robinson, 1847). In truth, the correct assumption is likely to lie somewhere in between.

At the start of the Industrial Revolution the rural areas, were called upon to supply the region's urban areas with fresh produce and goods like textiles (Atkinson, 1989; Floud and Harris, 1997; Rendall, 1990; Wrigley, 2004). Rural individuals were nowhere near as isolated as one might think. Farmers and tradesmen from small settlements routinely travelled to nearby market towns to sell their wares, purchase goods, and hire employees (Borsay, 1989; Harker, 1988). Men were not the only ones making these journeys. Women would also travel to markets and larger towns for shopping and cultural events. These travelers could therefore be exposed to the unsavoury living conditions, pollution, and disease, found in these larger towns.

Meanwhile, improvements were very slowly being made in most towns, to sanitation, air and water quality, and housing particularly in the middle of the 19th century (Hodgson, 1903; Linsley, 1992; Reid, 1845a, b; Robinson, 1847). The variety of foodstuffs seen in towns, but seldom in rural environments would lend itself to a healthy, well-balanced diet for those that could afford it (Gidney, 2009; Thirsk, 2006; Wilson, 2003). However, at the same time, living in a rural settlement allowed for the option of gathering wild plants and animals to increase the variety and cost effectiveness of food (Thirsk, 2006; Wilson, 2003). The price of food and clothing was becoming cheaper in towns due to the mass production of the Industrial and Agricultural Revolutions (Korzeniewicz, 1985). In the reasonably buoyant coal and shipping industry that dominated North and South Shields in the 18th and 19th centuries, much of the population would have likely been in a position to purchase a wholesome diet. Individuals steadily employed in North and South Shields may have been able to afford better quality housing than would have been expected, given their socioeconomic status, and their homes may have been better

quality than that inhabited by agricultural workers (Burnett, 1978; Green, 2006; Woodward, 1995).

5.4.4 Site Comparison Conclusions

The site comparisons undertaken in this section have met one of the aims of this thesis, namely to assess the prevalence of congenital defects between contemporary populations. The site comparisons have proved complex. Comparing the combined urban sites studied here with other urban locations around Britain demonstrated that the prevalence of congenital defects and "stress" indicators observed at North and South Shields were typical of patterns seen for towns of the 18th and 19th centuries. It also showed that the combined rural sites were similar to these same sites, implying that the individuals from rural contexts experienced similar levels of teratogens and other stressors in their environment as did individuals from urban contexts. When comparing the Quaker burial ground, North Shields with other Quaker sites from the south of Britain, higher prevalence rates of congenital defects and "stress" indicators were seen. This suggested that individuals from the other Quaker sites may have been protected from the detrimental living conditions of urban environments because of their socioeconomic status which allowed them access to better housing, food, and neighbourhood. Individuals from Wharram Percy showed an increase in congenital defect prevalence through time from the medieval to post-medieval periods, which suggests a decline in living conditions at Wharram Percy. To summarise, living conditions were likely to have played a role in the development of congenital defects but there was no observable division of conditions between urban and rural settlements.

5.5 The Urban/Rural Divide

From around the Roman period onwards in bioarchaeological research of Britain, perceived or even documented differential living conditions are explored to draw conclusions about what is seen in the skeletons of the individuals buried at

various types of locations. For example, Redfern and DeWitte (2011) found health worsened with the start of Roman urbanisation, Lewis *et al.* (1995) found higher frequencies of maxillary sinusitis in late medieval British urban populations compared to rural populations, and Judd and Roberts (1999) described differing patterns of long-bone trauma between urban and rural sites. The latter attributed the patterns observed to different occupations practised at the locations in medieval Britain. However, the post-medieval period seems to cast a shadow on the concept of visible health differences between urban and rural populations. As discussed previously (Section 2.3 and 5.2.2), detrimental living conditions were similar in both urban and rural environments in the post-medieval period. In popular thought, the post-medieval rural countryside has been seen as a peaceful idyll, contrasting with the coal-choked, overcrowded, diseased urban centres (Ellis, 2001a; Korzeniewicz, 1985; Robinson, 1847; Sharpe, 2000; Woods, 2003) albeit that historians are aware of the bias in these reports (see Section 5.6.5).

While this research into actual living conditions has done little to overturn that view of urban areas, it has also shown a rural landscape that is not so quiet. Industry branched out from urban centres to convenient points in the landscape such as rivers (water power), particularly in the North of England, leading to mills appearing in otherwise agricultural settings (Alred, 1997; Borsay, 1989; Jennings, 1967). This begs the question, of how rural is rural. The urban/rural distinction used for this study was based on population size and the main occupations of the inhabitants. Locations with a population over 5,000 and an economy based on occupations other than agriculture were considered to be urban for this study (Floud and Harris, 1997; Slack, 2000; Wrigley, 2004). Smaller settlements relying largely on agriculture, on the other hand, were deemed to be rural. Should a site be discounted from being rural if there is any sort of industrial practice being undertaken in the area? It may not be possible to answer this. Individuals from Fewston, considered to be rural for the purposes of this thesis due to its size and economy, frequently had the lowest prevalence rates of congenital defects. However, the parish of Fewston contained at least four textile mills starting in the 18th century (Alred, 1997). This raises the question of whether it was correct to

classify Fewston as a rural site. The trend of congenital defects is certainly what was expected for individuals from rural sites, but there was industrialisation occurring in the parish. In contrast, the frequency of "stress" indicators for individuals from Fewston was very similar to the rates seen at the other three sites, which was not expected for a rural location. The similar living conditions, as reflected in the data for skeletal "stress" indicators and congenital defects, and in the historical evidence associated with the sites in this study, may indicate that truly "rural" may not be so easy to locate for the post-medieval period.

5.6 Research Limitations

Anytime bioarchaeological research takes place, there will be limitations. Issues such as population representativeness and preservation are ever present when studying archaeological materials. When examining human remains specifically, the "osteological" paradox must always be considered as sometimes the lack of skeletal changes may hide the true prevalence of a disease. Cemeteries covering broad time spans may complicate interpretations of data, especially when trying to focus on living conditions within a limited time span. Additionally, comparisons of skeletal data to modern clinical information and historical documentation may not always be possible or appropriate due to bias.

5.6.1 Representative Burial Populations

The first limitation is the representativeness of the burial population. Not every individual who was living in a certain community was necessarily buried within that community's burial ground, as individuals may have emigrated from the area (and therefore been buried elsewhere) or been afforded differential burial rites (Roberts, 2009; Waldron, 1994; Wood *et al.*, 1992). The cemetery at North Shields was likely unrepresentative of the population of the town as it was a Quaker burial ground, excluding portions of the population based on religion. This exclusivity could have had an impact on the socioeconomic status (see Section 5.2.4) or the ages and sexes of the individuals buried there.

Burial populations may also be unrepresentative due to bias in excavation and taphonomy. In many instances, excavation of an entire cemetery is not possible; the portion excavated is then an imperfect subsample of an already biased sample of the population living within that community (Waldron, 1994). Excavating just one area may also bias the representativeness if different zones of a cemetery were divided by age, sex, or socioeconomic standing (Chamberlain, 2006; Saunders *et al.*, 1995; Waldron, 1994). The churchyards at both South Shields and Fewston were only partially excavated and therefore may have been biased in this way. Another bias, taphonomic processes, factors that affect the preservation of human remains after death, can have great effects on the skeletons of the very young, very old, or diseased individuals meaning these remains may be too poorly preserved for analysis or missing entirely from the burial population (Chamberlain, 2006; Nawrocki, 1995; Saunders *et al.*, 1995).

Preservation of skeletal remains varied greatly within and between the study sites, with Fewston having the highest proportion of poorly preserved skeletons. Additionally, skeletons were not always complete due to intercutting of other graves at all four sites. True prevalence rates for both congenital defects and "stress" indicators were calculated whenever possible in an effort to counteract the fact that some individuals or bone elements were too poorly preserved to be examined for a trait, but crude prevalence rates could not be avoided for many conditions. This raised the question during assessment of how intact or preserved a skeleton needs to be before it can be considered to not have at least one congenital defect. As a congenital defect could occur in any bone of the body, the ideal would be to have all bones present but in reality this is impossible to achieve in bioarchaeological studies. Perhaps the way forward would be to establish that a certain percentage of the bones need to be present, but as the vertebrae appear to be the most commonly affected region of the skeleton (Barnes, 1994; Kase, 2010; this study), it may be sensible to ensure that a percentage of the spine was always present. No logical consensus could be formed as to which individuals should not be included in the crude prevalence rates of this study so all skeletons labeled as discrete burials in the excavations reports were included.

5.6.2 The "Osteological Paradox"

The "osteological paradox" is a limitation to bioarchaeological studies as it states that bone lesions in a state of healing or ones that have healed may actually be a sign of relatively good health - 'better health makes for worse skeletons' (Wood *et al.*, 1992:356). It takes time for bone to show a visible reaction to an insult, but if a disease process or insult is short lived, no sign of "stress" will appear on the bone. Similarly, if the disease process kills the affected individual quickly, no sign will be left on the bone. An individual has to have lived long enough with the insult or disease before passing away for the bone to show any changes. A particularly "frail" population that is highly susceptible to disease may not have any osteological signs of poor health if they succumbed to the disease(s) quickly. This theory can be applied to congenital defects through the teratogenic model. Maternal exposure to a teratogen would have had to occur at the right gestational period and at the correct dose to elicit a physiological response, whilst simultaneously not being so detrimental to health as to cause the death of the expectant mother. Further, many congenital defects occur in only the soft tissues (ICBDSR, 2012; Office for National Statistics, 2010). Therefore, a teratogen may have been present at a location even though no evidence of a congenital defect can be seen on the skeleton.

Particularly of interest for the presence of "stress" indicators in this study are the co-morbidities reflecting various living conditions in the post-medieval period. Disease brought on by malnutrition (*e.g.* PH, CO, DEH) may exacerbate infections leading to a higher risk of death and a higher risk of that death occurring before bone changes occur that reflect those dietary deficiencies (Brickley and Ives, 2008). In return, dietary deficiencies lead to compromised immune responses and poor health (Christakos *et al.*, 2013; Verkerke *et al.*, 2012). Therefore, due to detrimental living conditions apparent in the 18th and 19th centuries, it may be possible that the prevalence rates for "stress" indicators seen here may actually be lower than they should have been due to premature deaths. The presence of bone lesions, meanwhile, indicates the individual was healthy enough to withstand the

insult long enough for the bone to respond. Therefore, as Wood *et al.* (1992:356) stated, 'a significant health advantage is reflected in a...higher frequency of skeletal lesions'. While the presence of "stress" indicators and other signs of disease are commonly used to distinguish poor health, it may actually indicate a slightly healthier population. The absence of these markers may indicate a population too frail to cope with onslaughts of physiological "stress" and disease. Fewston returning the highest prevalence rate for CO may actually indicate that that was the only population healthy enough to cope with disease or malnutrition. Individuals at the other locations may not have been able to survive long enough for bony changes to occur when faced with the same level of insult.

5.6.3 Broad Cemetery Dates

Another concern of bioarchaeological studies, but particularly in this one is length of time cemeteries were in use. When attempting to examine living conditions or health at multiple sites, similar dates of use make the interpretation of observed patterns easier. While all four of the study sites have dates that overlap during the 18th and/or 19th centuries, the populations from Fewston and Wharram Percy contained individuals buried as early as 1540 AD. The historical background provided in Section 2.3 attempted to cover the entire period of time that all four sites were in use, but that section demonstrated the extent of changes in industry, housing, and sanitation as well as exposure to diseases (plague epidemics in the 16th and 17th centuries and cholera in the 19th century) during the period. It therefore stands to reason that individuals from Fewston and Wharram Percy were exposed to a wider range of living conditions, whether good or bad, which may have affected the prevalence of congenital defects or "stress" indicators in the populations. If individuals with radiocarbon dates for the early post-medieval period from Wharram Percy are removed from the calculations for the crude prevalence rates of congenital defects and "stress" indicators both increase (from 43.33% to 45.10% for congenital defects and from 56.90% to 58.00% for "stress" indicators). Unfortunately, not all individuals from the post-medieval period at Wharram Percy were radiocarbon dated so individuals from the early period who

may have lowered the prevalence rates for defects could not all be excluded from this study. The numbers excluded here (nine individuals) were small, but this may show that over the several centuries of churchyard use in the post-medieval period at Fewston and Wharram Percy, living conditions may have deteriorated (or at least changed), which could have affected the comparisons made with cemeteries in use for shorter time spans like North and South Shields.

5.6.4 Comparisons to Modern Clinical Data

The next limitation in bioarchaeological research is that it is not always appropriate or possible to compare skeletal data with modern clinical data. This is seen particularly in the case of congenital defects. The vast majority of congenital defects recorded in this study are occult and asymptomatic meaning that when these defects are found in a clinical setting, it is usually as an incidental finding. Without radiographing an entire population, which is ethically frowned upon, it would not be possible to establish the actual prevalence rate of these occult defects (essentially creating a bias as to what anomalies are reported). Bioarchaeologists have the advantage of being able to easily macroscopically inspect the skeletons of populations for occult congenital defects, arriving at a more complete prevalence rate. For defects such as cleft palate which are symptomatic or easily visible from birth, modern rates are more likely to represent the true prevalence rate of a condition within a population and this data would therefore be comparable to bioarchaeological findings. For rates of individuals born in a population with at least one or more congenital defects, again the modern and bioarchaeological data are incompatible. Modern data includes soft tissue defects which cannot be seen in the skeletal record but alternatively, bioarchaeological data can contain skeletal defects that are either not seen in clinical settings or are not reported in clinical literature. Clinical reports of defects such as hypoplastic/aplastic transverse processes and aplastic ulnar styloid processes could not be found during this study. Essentially, bioarchaeological and modern clinical data do not compare like for like in the prevalence of congenital defects.

5.6.5 Historical Documentation

The final limitation is the use of historical documentation in bioarchaeological research. Mitchell (2012) states that much information can be gained through the use of historical documents but to avoid bias, primary sources should be preferentially used, texts should be read in the original language, and as many sources as possible should be utilised. However, there is still the risk of bias in historical documents. Individuals writing in the past, particularly before literacy became commonplace, were commonly men from the higher socioeconomic groups writing from their viewpoint. The experiences of lower class individuals or women may be missed or misrepresented (intentionally or otherwise) within these texts. Certainly for the earlier post-medieval period (17th century), many of the poorest inhabitants do not appear in written records of any type and therefore disappear.

This study consulted primary sources whenever possible in the original language (English) and attempted to draw from many different sources. However, bias could be seen within some of the primary sources. Writers of the 19th century seemed to have been more concerned with the morals of the poor than their health, commonly reporting that poor living conditions led to depraved morality and that the only way to lower crime rates was to improve living conditions (Chadwick, 1842; Robinson, 1847; Ellis, 2001a). The Poor Law Commissions after 1832 that resulted in reports on living conditions were undertaken with a heavy moral underpinning (Johnson, 1993). It was argued at the time that the reports found what they had set out to find, were based on leading questions, some portions were written prior to visitations, and that, consequently, the reports may have emphasised the squalor and worst scenarios in order to instigate and justify rapid reform.

5.6.6 Research Limitations Conclusions

This section has demonstrated that there are limitations when undertaking bioarchaeological research. Whenever drawing conclusions is based on

bioarchaeological assessments, these limitations must always be considered and account taken for the uncertainty that may therefore exist. However, the limitations in bioarchaeology are not as great as this section may make it sound as every study using skeletal remains will have the same limitations meaning comparisons between data can still be made. Despite any limitations, bioarchaeological research such as this study is still vital in understanding health and disease in the past (Wood *et al.*, 1992).

5.7 Conclusion

This chapter provided a review of the results found for both congenital defects and "stress" indicators, originally detailed in Chapter 4, demonstrating that the study hypothesis of congenital defects occurring at higher prevalence rates in urban locations could not be supported. Similarly detrimental living conditions between the two contexts, migration, socioeconomic status, and the aetiology of congenital defects were all explored in connection with the data as possibilities explaining why the hypothesis had to be rejected. The data was then compared to contemporaneous populations to see how representative the results of this study were for the post-medieval period. Lastly, the limitations intrinsic to bioarchaeological research were discussed in relation to this study.

Through the sections detailed above, this chapter has demonstrated that despite the lack of statistically significant results and the limitations inherent in bioarchaeological research, conclusions about past living conditions, congenital defects, and "stress" indicators may still be drawn. Living conditions in both the urban and rural sites studied in this research were detrimental to health, although high population densities in urban areas may have exacerbated exposure to poor conditions. Aspects of housing, diet, occupation, sanitation, and infectious disease all paint pictures of environments that could lead to the development of congenital defects. The location of intensive, polluting industries in rural settings of this period blur the lines of where urban stops and rural begins in studies such as this

attempting to differentiate between health and living conditions in the two contexts. As the pattern of congenital defects and "stress" indicators were similar in this study, congenital defects may be a useful indicator of detrimental living conditions. However, due to the genetic component of these defects, they are not a particularly good measure on their own. By combining bioarchaeological assessment of congenital defects with "stress" indicators and historical/archaeological evidence, it has been possible to provide a deeper understanding of the health of past populations.

CHAPTER 6
CONCLUSION

'Through our work new insights must be gained into the hidden detail of ordinary lives...In so doing we need to make the stories that we tell more interesting and relevant.'
(Symonds, 2006:240)

6.1 Introduction

This thesis has examined the link between the presence of congenital defects and poor living conditions in urban and rural populations from 18th and 19th century Northeast England. Studies such as this may have potential applications in combating the development of congenital defects today, especially in industrialising nations where living conditions are not dissimilar to that experienced in the post-medieval urban populations examined here (WHO, 2012). This chapter will review the results of this study in relation to the hypothesis, aims, and objectives. Additionally, limitations of this study as well as recommendations for future improvements and work will be discussed.

6.2 Review of the Thesis

In Chapter 1, the reasons for undertaking this study were set out and definitions relevant to the study of congenital defects were given. Congenital defects are present worldwide and can be found today and throughout history. Evidence of congenital defects in the past can be found in documentary resources as well as in skeletal remains (Barnes, 1994; Bašić *et al.*, 2012; Bates, 2000, 2005; Blumberg, 2009; Keenleyside, 2012a; Lieverse *et al.*, 2012; Pany and Teschler-Nicola, 2007; Paré, 1573). Having an insight into living conditions in the past that may have caused these anomalies may be useful in combating their development in

modern populations. The post-medieval period in England was chosen for this study due to it being under-represented in the archaeological and bioarchaeological literature (Cherryson *et al.*, 2012; Matthews, 1999; Petts and Gerrard, 2006). Additionally, the 18th and 19th centuries were marked by an expansion in urban living and the development of industrialisation that appear to have caused a decline in the standard of living (Briggs, 1990; Brimblecombe, 1978, 1987; Ellis, 2001a; Holt and Rosser, 1990; Roberts and Cox, 2003). Many of the health problems seen in this 2nd epidemiological transition of the past can still be seen in modern populations within industrialising nations (Dellicour *et al.*, 2013; McDade and Adair, 2001; McMichael, 1999; Oliveira *et al.*, 2011; Peters, 1999; WHO, 2012).

Chapter 2 was a five-part literature review. Section 2.2 discussed clinical research on the aetiology of congenital defects, focusing largely on environmental factors. This was followed by Section 2.3, a review of historical literature on the living conditions seen specifically at the study sites. Topics included housing, sanitation, air and water pollution, occupation, diet, and mortality. Section 2.4 described the congenital defects observed in this study using clinical and bioarchaeological sources. For each condition, information was provided on its aetiology, rates of occurrence in modern populations, and clinical symptoms as available. The same treatment was given to "stress" indicators in Section 2.5. The chapter concluded with Section 2.6 looking at the bioarchaeological literature for evidence of congenital defects. The section started with a review of the five multiple population studies known to the author where congenital defects were compared across temporal or geographical regions. Next, single population studies and case reports documenting congenital defects in Britain in the 18th and 19th centuries were examined. A variety of defects were recorded, but there was a dearth of site reports for cemeteries of the period.

Chapter 3 described the materials and methods used for this study. First each of the four study sites was briefly introduced and historical 19th century maps were provided showing the location of each cemetery. Methods for sexing and aging of each skeleton were then detailed. This was followed by an explanation as

to how each congenital defect and "stress" indicator was diagnosed in the skeletal populations within this study.

Chapter 4 presented the data on congenital defects and "stress" indicators observed in the skeletal populations obtained using the methods from Chapter 3. Each trait was presented individually and in combined groupings. Fisher's exact test or a t test was completed for each condition where appropriate and any statistically significant results were highlighted. Photographs of the congenital defects and "stress" indicators taken during assessment of the skeletal remains were provided.

Chapter 5 discussed the data that had been provided in Chapter 4 in relation to the study hypothesis. References to the historical and clinical literature discussed in Chapter 2 were made in an attempt to explain the patterns observed in the congenital defects and "stress" indicators. The study sites were then compared to multiple contemporaneous sites to explore patterns on a broader scale within Britain and determine the representativeness of the data produced in this study. Additionally, late medieval individuals from Wharram Percy were compared to the post-medieval individuals studied in this thesis to look for changes over time within a location. Finally, the limitations of bioarchaeological studies and their potential effects on this study's results were discussed. This chapter determined that by combining congenital defect and "stress" indicator studies with historical evidence, an understanding of past living conditions can be obtained.

6.3 Hypothesis

In Section 1.3, it was hypothesised that congenital defects were more common in urban populations rather than populations from rural locations in 18th and 19th century Northeast England. This would be as a result of living conditions that placed health at risk and were associated with urban, industrial areas of the post-medieval period. This hypothesis was formed based on studies by Sture (2001) who found higher prevalence rates of congenital defects in urban medieval populations from England and Kase (2010) who found higher prevalence rates in

late and post-medieval urban populations from Britain. Due to the results presented in this thesis, the hypothesis could not be supported as there were no statistically significant differences in the frequency of congenital defects between urban and rural sites for the presence of combined or individual congenital defects. Furthermore, individuals from the two rural sites had the highest (Wharram Percy) and lowest (Fewston) prevalence rates for combined congenital defects. In order to have validated the hypothesis, the rural sites would have had the two lowest defect rates.

There are several possible explanations for this lack of statistical significance, the first being that living conditions in urban and rural settlements were equally detrimental to health. As discussed in Sections 2.3 and 5.2.2, teratogenic and other harmful factors were found within both contexts in the form of infectious disease, air and water pollution, and an inadequate diet. The second possibility is the effect of migration (see Section 5.2.3). If indeed it is true that congenital defects were less common in rural areas due to healthier living conditions, individuals migrating from the countryside to towns in search of employment (as was common in the 18th and 19th centuries) could decrease the overall prevalence rates for congenital defects seen within an urban cemetery. The third possibility is the effect of socioeconomic status on congenital defect development (see Section 5.2.4). Assigning socioeconomic status to individuals based on parish records is problematic for this period so this possibility could not be explored fully. However, the parishioners from Wharram Percy could be ascribed to a low socioeconomic status and the Quakers from North Shields to the middle classes. As Section 4.3 showed, compared to North Shields, parishioners from Wharram Percy had a higher frequency of congenital effects as would be expected of poorer individuals (see Sections 1.1.2.C and 1.2.2). The fourth, and final, possibility is the role of genetics in the development of congenital defects (see Section 5.2.5). As both environmental and genetic factors can lead to the development of congenital defects, it can be difficult to attribute the presence of congenital defects in the past to just one cause. The answer for why the data could not support the study hypothesis was likely a combination of the factors discussed

above. Without continued research into the frequency of congenital defects in the post-medieval period, it may not be possible to conclusively state why this study has demonstrated that there was no difference in the frequencies of congenital defects between urban and rural sites in Northeast England in the 18th and 19th centuries.

6.4 Aims and Objectives

6.4.1 Aims

In order to determine the validity of the hypothesis three research aims were laid out in Section 1.3. These aims are reproduced here and were:

- To assess the impact of teratogenic agents on 18th and 19th century populations in Northeast England;
- To consider the importance of settlement type (urban/rural) on the frequencies of congenital defects; and
- To compare the prevalence of congenital defects between contemporaneous populations in the two contexts.

Each of these aims will be discussed in turn below.

The first aim was to assess the impact of teratogenic agents such as infectious disease, malnutrition, heavy metals, and hazardous gases on populations as they were present at the study sites in the 18th and 19th centuries (Section 2.3). Infectious disease spread rapidly through the overcrowded unsanitary towns (Hodgson, 1903; Horsley, 1971; Openshaw, 1978; Wrightson, 2009) but exposure to unsanitary conditions, other humans, and livestock in rural locations meant individuals there were exposed to infections too, although not likely on the same scale (Harker, 1988; Hey, 1986). A limited diet with little access to meat or fresh vegetables was common in the period at both urban and rural locations (Drummond and Wilbraham, 1957; Hey, 1986; Ketabgian, 2007; Wilson, 2003) while

heavy metals and poisonous gases were released into the atmosphere of urban and rural areas by burning coal, both in the home and in industries, although again, the effects would likely have been exacerbated by high population densities in urban locations (Alred, 1997; Atkinson, 1989; Campbell, 1968; Hodgson, 1903; Jennings, 1967; Linsley, 1992; Moffat and Rosie, 2005; Reid, 1845a, b; Thornborrow, 1971a, 1988; Warren, 1980). While it is known these teratogens existed, it has unfortunately not been possible to fully assess the impact of these agents on the past populations studied due to the aetiology of congenital defects. Determining the teratogenic cause of a specific defect is extremely difficult in living populations (Hill *et al.* 2008; Holmes 2011; Li *et al.* 2013) and in bioarchaeological populations it is impossible. Many teratogens cause the same skeletal changes and, without interviewing the individual's mother, it cannot be conclusively stated which particular teratogens she may have come into contact with whilst pregnant. It can be said that teratogens may have led to the presence of congenital defects in these past populations, but nothing can be determined about which specific poor living conditions resulted in a particular defect being seen in any given individual. Additionally, congenital defects may be caused by genetic as well as environmental factors (Barnes, 1994; Fan *et al.*, 2013; Schoenwolf *et al.*, 2009; WHO, 2012). A genetic inheritance or mutation may cause defects, but genetics can also control the expression of a defect caused by a teratogen (Holmes, 2011; Schoenwolf *et al.*, 2009). Therefore, when attempting to ascertain why congenital defects are seen in past populations, both intrinsic and extrinsic factors should be considered in the discussion. While both teratogenic agents and congenital defects were present in the past populations studied here, there is no method available to quantify the impact of certain teratogens on these individuals. Instead, the conclusion can be made that the poor living conditions seen at the study sites (Section 2.3) almost certainly contributed to the development of congenital defects but the level of impact specific teratogenic factors had on health in the past is unknown.

The second aim was to assess the importance of settlement type on the frequencies of congenital defects. As the hypothesis was that congenital defects would be more common in individuals from urban contexts, it was assumed urban

settlements would have a greater impact on the frequencies of defects in their populations than rural ones. The data, however, show that there was no statistically significant difference in the prevalence rates for congenital defects occurring in individuals from either urban or rural contexts (see Sections 4.3 and 5.2.1). The two rural sites, Wharram Percy and Fewston, had the highest and lowest prevalence rates of combined congenital defects respective, with the frequencies at the urban sites of North and South Shields in between Wharram Percy and Fewston. These patterns imply settlement type was not important to the recorded frequency of congenital defects. This was further reinforced when the data were compared to five other urban sites across Britain (Section 5.4.1). The prevalence rates for both the urban and rural sites were similar to those found at the comparison sites.

Part of this conclusion may be explained by a lack of a strict dichotomy in settlement types of the period (see Section 5.5). For this research, as was detailed in Section 1.4, an urban settlement was defined as having a population over 5,000 and an economy based on industries other than agriculture (Floud and Harris, 1997; Slack, 2000; Wrigley, 2004). Conversely, rural sites had smaller populations and an economy based largely on agriculture. However, there can be locations that rely mainly on agricultural practices but also have heavy industry. As Langton (2000:486) writes, 'Splitting places into urban and rural, implying that each category is uniform and remained essentially static through history, is neat, convenient and conventional; but it is impossible to decide into which category many of the most dynamic places of this period fit.' North Yorkshire in this period could certainly be considered "dynamic" with the rise of the textile and mining industries (Alred, 1997; Atkinson, 1989; Hey, 1986; Jennings, 1967; Singleton, 1970). Fewston is an example of a location that may not "fit" easily into a dichotomous labeling system of urban versus rural. The parish, while having agriculture as the main employment and a population under 2,200 at its highest, contained four textile mills that started operations from the 18th century. Industrial practices in rural settlements were not unheard of in this period, and this was certainly not limited to just Fewston (Alred, 1997; Borsay, 1989). However, the presence of these industries and their associated pollution are likely to have affected the living conditions of the inhabitants of these

rural communities, making them not quite "urban" but not quite "rural." It seems fair to say that settlement type did not greatly affect the frequencies of congenital defects seen in this study and this may be due to a lack of distinction between the environments of urban and rural locations.

The third aim was to assess the prevalence of congenital defects between contemporaneous populations from urban and rural contexts. Sites were chosen for comparison to give a wide range of socioeconomic statuses and locations, although the "publishing bias" led to more than one site being located in London (see Section 2.6.3). Sites chosen for comparison were St Martin's-in -the-Bull-Ring from Birmingham, St Peter's from Barton-upon-Humber, the Cross Bones burial ground, Southwark, Chelsea Old Church, and St George's, Bloomsbury, all from London, and three Quaker burial grounds from Kingston-upon-Thames, King's Lynn, and St Ives. As was discussed in Section 5.4, little difference was found between data from contemporaneous comparison populations and the study sites. When the Quaker burial ground at North Shields was compared to the other Quaker sites, some statistically significant results were found (Section 5.4.2). Individuals from North Shields had a higher prevalence rate for combined and/or individual defects compared to individuals from either Kingston-upon-Thames or King's Lynn but prevalence rates were similar between North Shields and St Ives. It was concluded that the living conditions at North Shields were worse than those experienced at Kingston-upon-Thames or King's Lynn, particularly in terms of overcrowding and air pollution.

6.4.2 Objectives

The objectives for this thesis, as given in Section 1.3, were:

- To generate a population study of congenital defects in 18th and 19th century Northeast England; and
- To provide a better understanding of the effects of health and living environment on the occurrence of congenital defects.

This thesis has provided a detailed population study of congenital defects in 18th and 19th century Northeast England, with congenital defects recorded for four different populations of individuals from two urban and two rural locations. The results of this study are in Chapter 4 as well as Appendices B through E. Sex and age information was provided for each individual when able and as much detail as possible was included in the description of the observed congenital defects. This was done to make these data useful for comparison with other sites of this and other time periods, not only for this research but also for any future comparison of work by others. As few multiple population studies of this kind have been undertaken in the past (*i.e.* Barnes, 1994; Kase, 2010; Masnicová and Beňuš, 2003; Murphy, 2000; Sture, 2001), with only Sture and Kase looking at individuals in Britain, this thesis adds greatly to the knowledge of the distribution of congenital defects and their meaning.

This thesis has met the second objective by producing an improved understanding of the impact of individuals' living environment on their health and the occurrence of congenital defects. This research has demonstrated that known teratogens existed in the living environments of both the urban and rural site locations (see Sections 2.2, 2.3, and 5.2.1). Air and water pollution, infectious disease, and poor nutrition can all lead to the development of congenital defects, and were all present in the 18th and 19th centuries at the study sites. Since these teratogenic factors were present at both urban and rural sites and similar rates of congenital defects occurred at all four sites, there may be a link between poor living conditions and the presence of congenital defects in past populations. "Stress" indicators were used in an effort to further determine which sites had poor living conditions. The data followed the same pattern as that seen for congenital defects, but the frequencies were much more similar between sites for "stress" indicators than they were for congenital defects. This finding suggests that all four sites had similar levels of environmental stress. As discussed previously (Sections 5.2.5 and 6.4.1), specific teratogens cannot be linked to specific congenital defects in skeletal remains, making it difficult to quantify the level of impact particular extrinsic factors had. However, since the same pattern of prevalence of "stress" indicators (caused

by “environmental” factors) followed that of the data for congenital defects, it should be acceptable to say that there is a link between the presence of congenital defects and poor living conditions.

6.5 Research Limitations

As was discussed in Section 5.6, there are limitations inherent in any bioarchaeological research and this study was no exception. The factors that may have particularly affected the data of this study are summarised here and include preservation (taphonomic processes), the “osteological paradox,” an inability to compare to modern clinical data, and differing cemetery dates.

Poor preservation can limit the ability to analyse skeletons for congenital defects or “stress” indicators. True prevalence rates were calculated whenever possible but crude prevalence rates could not be avoided for some conditions. This means that skeletons too poorly preserved or too fragmentary to observe for congenital defects or “stress” indicators were still included in some of the calculations, which may have correspondingly affected the frequencies.

The presence of congenital defects or, particularly, “stress” indicators were interpreted here as signs of living conditions that were detrimental to health. However, the “osteological paradox”, in which bone lesions in a healing or healed state may indicate better health than the absence of lesions (Wood *et al.*, 1992), can alter the way the data of this study is interpreted. While it can still mean that these conditions were caused by poor living conditions, it is much more problematic to interpret a higher prevalence of these conditions within a population at one location as meaning the health of those individuals was worse than for individuals at another location.

The occult and asymptomatic nature of the majority of the congenital defects recorded in this study makes it difficult to compare this data to that from modern clinical studies. Asymptomatic defects are generally only found in clinical

settings surreptitiously, meaning the actual prevalence of that defect in modern populations cannot be known. Additionally, clinicians are able to record soft tissue anomalies that cannot be seen skeletally while bioarchaeologists are able to record skeletal defects that are not seen, at least commonly, in modern populations. This means the overall rates of congenital defects in modern and past populations are not directly comparable.

Finally, while the periods the cemeteries were in use at all four study sites overlapped in the 19th century, the cemeteries from the rural sites of Fewston and Wharram Percy were in use much earlier than the urban sites of North and South Shields. Some of the individuals included in this study from the rural sites were buried as early as the 16th century compared to the 18th and 19th centuries at North and South Shields. As living conditions did not remain static, especially at the start of the Industrial Revolution, the individuals buried in the earlier post-medieval period may have experienced different living conditions (for better or worse) than those buried in the later period. This may have impacted on the frequencies reported in this study.

Every bioarchaeological study has limitations but that does not mean the data obtained is of little value. These limitations should be kept in mind when conclusions are being drawn about health in past populations but as every bioarchaeological study will have similar weaknesses, comparisons between studies are still possible. Research of this nature is extremely useful in gaining information about health and disease in the past as well as furthering knowledge about living conditions when bioarchaeological data is combined with historical documentation.

6.6 Further Work

This thesis has helped to improve the understanding of the impact the environment had on congenital defects in the post-medieval period. However, there are still many avenues available for future investigation. This section will discuss the potential work that may be undertaken to improve bioarchaeological

knowledge of congenital defects. Firstly, the methods in which congenital defects have been presented are examined with reference to improvements that can be made in future reports. Next, the recommendation that charnel deposits are examined for congenital defects in future excavations is made. Defects found in these remains may enhance the understanding of congenital anomalies at any given site. Finally, potential areas of further work in the areas of post-medieval bioarchaeology and multiple populations studies are addressed. In order to enhance the knowledge of congenital defects in Britain in the past, focus should be placed on increased bioarchaeological evaluation of post-medieval skeletons, particularly those from rural contexts, and more research in which multiple sites or time periods are examined.

6.6.1 Reporting of Congenital Defects

Reading skeletal reports, one question keeps arising: where is the evidence for congenital defects? Firstly, it is a matter of consistency. While many reports will have a section on congenital defects, not all of the defects may actually be reported there. Congenital defects seem to "find their way" into the non-metrics traits sections of reports very frequently, particularly os acromiale, but others such as cleft neural arch, sacralization, and supernumerary vertebrae can also occasionally be found there (*e.g.* Adams and Colls, 2007; Clough and Loe, 2007; WORD Database, 2013a, b). Spondylolysis, and less frequently os acromiale, can also be commonly found in the trauma sections of reports. This is not surprising given the somewhat questionable aetiology of spondylolysis (congenital weakness and/or trauma). This "hide and seek" can lead to congenital defects being overlooked in data by readers attempting to gain insight into their presence within a population. To improve the situation, a standard division of traits/pathologies should be adopted with reference to congenital defects, perhaps taking Barnes's works (1994, 2012a) as an exhaustive list as to what constitutes a congenital defect.

Secondly, the matter of reporting congenital defects, *per se*, is a problem. In skeletal data where they are not reported, the question is: are they really not present? While individually rare, congenital defects would be expected to be seen

in any sizeable population. Assuming they are present, why are they not included in the report? Are they not thought important enough to include, or is the researcher examining the skeletons not noticing them? For example, lumbarization of the T12 vertebra appeared to be quite common in this study (11.21% CPR for all sites combined) but is extremely underreported (pers. obs., Caffell, pers. comm.). However, because this defect would cause no problems in the affected individual (Barnes, 1994), is it not reported due to the insignificance of the clinical manifestations? Lumbarization of T12 was recorded at the Cross Bones cemetery in London as is shown in the WORD Database of raw data but no mention of this defect could be found in the published report (Brickley *et al.*, 1999; WORD Database, 2013b). As minor congenital defects can serve as a proxy for more major congenital defects that may not be seen archaeologically due to cultural practices or taphonomic processes (see Section 3.3) and for congenital defects completely missing from the skeletal record as they only affect soft tissues (Barnes, 1994; Chamberlain, 2006; Cherryson *et al.*, 2012; Gittings, 1984; ICBDSR, 2012; Lewis, 2007; Office for National Statistics, 2010; Pitre and Lovell, 2010; Tocheri, 2005; Waldron, 1994; Wood *et al.*, 1992), all defects should be considered to be worth recording when assessing the palaeopathology of skeletal populations.

Another reason for their absence in reports may be that the defects are not being observed. For example, lumbarization of T12 is diagnosed through very subtle changes to the lowest thoracic vertebra (articular facets rotated up to 90^{o} to take on the appearance of lumbar facets, and aplastic/hypoplastic costal facets) and complimentary changes to the first lumbar vertebra. If someone is not trained to know what a normal T12 should look like, the condition would be incredibly easy to overlook. At the same time, someone properly trained but, in a rush due to deadlines, could just as easily fail to recognise it. It is not possible to know the answers to the questions raised here, but if bioarchaeologists are properly trained to identify congenital defects and are assured of their value in palaeopathology, these issues may be avoided in the future.

Additionally, the matter in which the information is reported should be improved. If basic specific information such as which congenital defects are present, crude prevalence rates, and (preferably) true prevalence rates are not included in skeletal reports, the fact that one or more congenital defects were found is of little value. This is because the data cannot subsequently be compared with other sites to gain insight into congenital defect frequency through time or within a region. Reports reviewed for this study provided the percentage or number of individuals affected by congenital defects in certain regions of the body, but not the actual details of the defects present (*e.g.* Start and Kirk, 1998; Waldron and Rodwell, 2007). Every skeletal report should include as much information as possible on congenital defects as is allowed by the size constraints of the publication, but at the very least should contain the defects observed and the number of individuals affected. To further the information available to other researchers, especially when space is at a premium in printed materials, skeletal recording forms could be archived either in hard copy or electronic form. Databases created to contain the information obtained from the skeletal assessments could be made available online or through a DVD/CD included with the published report.

Finally, terminology and diagnostic criteria should be standardised so the information reported can be easily understood and comparisons can be made. A uniform vocabulary should perhaps be based on Barnes (1994, 2012a) and a thorough evaluation of current medical literature. A common example found for differential use of terms is Klippel-Feil syndrome and block vertebrae. In its original description in the medical literature, Klippel-Feil syndrome referred to a congenital fusion of cervical vertebrae, a shortening of the neck, and a webbed neck (a skin fold running from the neck to the shoulder) (Barnes, 1994, 2012a; Ozonoff, 1995; Pany and Teschler-Nicola, 2007; Resnick, 1995b; Samartzis *et al.*, 2008; Silva and Ferreira, 2008). Since the original description, in both clinical medicine and palaeopathology, the term now means congenital fusion of vertebrae occurring anywhere in the spine (Olufemi Adeleye and Olusola Akinyemi, 2010; Silva and Ferreira, 2008). Often in palaeopathological literature, this term is only used for fusion in the cervical vertebrae and this research continued that trend (*e.g.* Brickley

et al., 2006b; Waldron and Rodwell, 2007). Given that the original description includes a soft tissue change, something which cannot ever be reported using skeletal material, perhaps "block vertebrae" or "congenitally fused vertebrae" for fusion occurring anywhere in the spine, would be the better terminology to use in bioarchaeological research. Additionally, some terminology in use today may be inappropriate for all manifestations of the condition. A shift in the caudal direction at the thoracolumbar border is typically referred to simply as a lumbar rib since these are the most noticeable change in this defect. However, the bioarchaeological assessment of this study found that not all caudal shifts at that location resulted in the presence of lumbar ribs (see Section 4.3.8.C and Appendices B through E), instead changes were seen in the apophyseal facets only. Therefore, a better use of terms would be a caudal shift or perhaps "thoracization" to improve the representativeness of the condition's terminology.

Regarding misdiagnosis through the misuse of terminology, the most frequent found in palaeopathological reports is that of spina bifida occulta. For some reason, spina bifida occulta has come to mean clefting of the neural arches of S1 to S5 (*e.g.* Adams and Colls, 2007; Boston *et al.*, 2009; Boyle *et al.*, 2005; Brickley *et al.*, 1999; McCarthy *et al.*, 2009; Miles *et al.*, 2008b; Waldron and Rodwell, 2007). As discussed in Section 2.4.4, spina bifida occulta can occur anywhere in the spine, although it is most commonly found in the sacrum and lower lumbar vertebrae. It represents a delay in posterior fusion of the bone at the midpoint due to an obstruction caused by soft tissues. Unless there is evidence of flaring of the bony arch at the cleft, showing soft tissue had caused an obstruction and pushed the bony arch away from the normal location, these defects should be diagnosed as cleft neural arch and not spina bifida occulta. The term cleft neural arch relays a less serious connotation that is more appropriate to this defect than that of spina bifida occulta: cleft neural arch does not have clinical symptoms but spina bifida occulta can result in neurological defects (Barnes, 1994; Ozonoff, 1995). Additionally, clefting seen at S1, S4, or S5 has been noted to be nearly universal so these occurrences should not be recorded as defects unless there is some obvious hypoplasia or aplasia on any portion of the neural arch.

Having explained the current situation of how congenital defects are reported in the bioarchaeological literature, the recommendation is that to better improve future comparative analyses and research, more attention needs to be paid to congenital defects, they need to be reported consistently, and they should not be overlooked in favour of other normal and abnormal variant lesions in skeletal remains.

6.6.2 Charnel

Charnel or disarticulated remains are often overlooked in bioarchaeological recording and generally for good reasons (McKinley, 2004). Usually there is a limited budget and time frame and understandably preference is given to articulated individuals over charnel as more information is likely to be gained from the intact skeletons. However, the analysis of charnel can be very informative (Ortner, 2011; Turner, 1993; Willey *et al.*, 1997) and the current research has shown that diagnoses of congenital defects can be made in disarticulated remains (Section 4.3.20). Congenital defects seen in skeletal remains often affect just one bone, only seldom being seen as syndromes involving a wide range of bones of the skeleton (Barnes, 2012b; see also Section 2.4), allowing a diagnosis to be made with just the presence of one bone. This is unlike many other disease processes observed bioarchaeologically such as tuberculosis, leprosy, syphilis, rheumatoid arthritis, and scurvy where multiple regions of the skeleton need to be present to make a diagnosis. While isolated bones cannot provide much information on their own as they lack vital information such as age, sex, and associated defects or diseases, they may be incorporated into counts of true prevalence rates for congenital defects to give an even broader understanding of their occurrence in a particular population. It is therefore recommended that charnel remains are examined for congenital defects whenever it is possible to do so as they may give a more realistic indication of the prevalence of some defects within that population.

6.6.3 Post-Medieval Sites

There is currently an under-representation of English post-medieval cemeteries recorded bioarchaeologically in the literature as was discussed in Section 2.6.3. These sentiments are echoed by Cherryson *et al.* (2012) and Petts and Gerrard (2006). The dearth of post-medieval sites is especially true for rural sites from this period. When undertaking the site comparison portion of this research, no suitable rural site could be found for comparison. The anthology of post-medieval burials published by Cherryson *et al.* (2012) demonstrated that archaeological surveys, if not excavations, have taken place at a large number of post-medieval cemeteries spread across Britain. However, the information gained from these exercises is either very scanty (full bioarchaeological examination did not take place as part of the excavation), unavailable due to the work not being published, or both. Vast amounts of data are potentially available but are useless unless it is made available either online or through a publication.

Currently, the majority of site information that is available for skeletal collections of the post-medieval period comes from London and the south of England in general, mostly because excavations of post-medieval cemeteries are more common in London due to demand for building on unused land, but also because there seems to be more of a willingness to publish the reports in that region. To better understand the impact of congenital defects on past populations of this time period, and to fill gaps in historical records in the post-medieval period, it is recommended that future studies should bioarchaeologically analyse more post-medieval cemeteries, particularly those in rural locations, and allow for these reports to be disseminated.

6.6.4 Multiple Population Studies

More multiple population studies need to be undertaken to gain a better understanding of the occurrence of congenital defects across time and space and to ascertain the meanings behind these patterns. Case reports or individual site reports are important for disseminating information about the presence of

a/multiple congenital defect(s), but a better understanding of the reason congenital defects occur when and where they do can only be gained through multiple site studies. Prevalence rates need to be compared and examined for patterns that may show differences in living conditions, cultural patterns, or genetic relatedness. Furthermore, other regions of Britain in the post-medieval period should be examined to see if the lack of an urban/rural divide holds true in other areas and to allow for comparisons geographically (*e.g.* are prevalence rates in the North different from those seen in the South?). Other time periods should also be investigated to eventually produce evidence of congenital defects across the centuries allowing for changes through time to be studied alongside sociocultural changes. Basically, the more data produced, the better the understanding of these often overlooked anomalies will become.

6.7 Concluding Remarks

This final chapter has established the purpose of this study through examination of the hypothesis, aims, and objectives. Based on previous work by Sture (2001) and Kase (2010) revealing higher prevalence rates of congenital defects in urban populations of the medieval and post-medieval periods in Britain due to the unsanitary conditions found in urban environments, hypothesis of this study was formulated. The study hypothesis was that higher frequencies of congenital defects would be found in urban populations of 18th and 19th century Northeast England when compared to their rural counterparts as this period was known for its decrease in living conditions associated with urbanisation and industrialisation. The data of this study (see Chapter 4 and Section 5.2) proved that the hypothesis could not be supported. However, while investigating the hypothesis, this thesis was able to assess the impact of teratogenic agents on these populations, to consider the importance of settlement type on the frequencies of congenital defects, and to compare this data with other contemporaneous populations. It was also able to generate a population study of congenital defects in 18th and 19th century Northeast England and to provide a better understanding of

the effects of living conditions on the occurrence of these defects. This chapter also included a discussion on the limitations of bioarchaeological studies and offered recommendations for future potential work to be undertaken. Congenital defects should be investigated further in the future, and to better improve future comparative analyses, these defects need to be given more attention, be reported in a consistent manner, and given the same attention as other aspects of palaeopathology in bioarchaeological studies.

As Sharpe explains, 'The long-running "standard of living debate" for the Industrial Revolution period suffers from befuddled attempts to compare dissimilar and incomplete types of evidence and to construct a national picture from a myriad of regional differences' (2000:509-510). This thesis challenged this idea by focusing on one region, Northeast England, and comparing similar types of evidence. The image of congenital defects and past living conditions presented in this study through examination of human skeletal remains as well as clinical and historical documentation shows a clear link between the occurrence of congenital defects and poor living environments in the 18th and 19th centuries. This link is not limited to the urban populations, as was proposed by the study hypothesis, but can also be seen in rural areas since these settlements were not immune from poor living conditions. Congenital defects on their own unfortunately do not seem to serve as indicators of past health due to their multiple aetiologies. However, combined with data on "stress" indicators and historical/archaeological evidence, congenital defects may be able to provide a deeper understanding of the health of past populations.

The best way to conclude this thesis is with a farewell to the region but making sure not to forget the individuals who lived through this turbulent, dynamic, exciting, and hazardous period in Northeast England and how the living conditions of the period affected their health and lives:

"Hookey Walker's Farewell to Shields"
by William Brockie, 1852

'Farewell to Shields, the filthiest place
On old Northumbria's dirty face,
The coal-hole of the British nation,
The fag-end of the whole creation,
The jakes of Newcastle-upon-Tyne
The banquet house of dogs and fleas
And human vermin worse than these;
A mass of houses-not a town-
On heaps of cinders squatted down,
Close to the river's oozy edge,
Like moulting chickens behind a hedge;
Huge ballast heaps, from London brought,
And here, like churchyard rubbish, shot. . .
And, when it rains or thaws, a flood
Of sticky, stinking, coal-black mud,
Oft ankle-deep, in Claypath Lane,
Making the use of blacking vain. . .
Steamtugs, whose smoke beclouds the river;
Chimneys, forth vomiting for ever
All sorts of gas, to taint the air,
And drive the farmers to despair. . .
Streets-if the name can be applied
To dingy lanes not ten feet wide-
Bordered by wretched tenements
Let to poor devils at high rent.
If brutes have souls, as some pretend,
And after death to Hades wend,
And learn to speak; I do expect
'Twill be in the Shields dialect.
Farewell to Shields' I shout again;
A long and glad farewell! Amen!
I never liked the place, nor did
The place like me: but God forbid
I should hear witness false against it;
I have writ the truth, and here attest it.'
(Hood Coulthard, 1960:36)

CONGENITAL DEFECTS IN 18TH AND 19TH CENTURY POPULATIONS FROM RURAL AND URBAN NORTHEAST ENGLAND

VOLUME II

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Thesis Submitted for the Degree of Doctor of Philosophy

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VOLUME II:

APPENDIX A
BLANK SKELETAL RECORDING FORMS

List of Abbreviations for the Blank Recording Forms:

#	number
c	deciduous canine
C	permanent canine
CO	cribra orbitalia
i1	first deciduous incisor
I1	first permanent incisor
i2	second deciduous incisor
I2	second permanent incisor
m1	first deciduous molar
M1	first permanent molar
m2	second deciduous molar
M2	second permanent molar
M3	third permanent molar
obs	observable
PM1	first permanent premolar
PM2	second permanent premolar

Site:_____ Skel #:_____ Date:_____

ADULT SEX ASSESSMENT

SKULL	Male	?Male	?	?Female	Female	Unable
Supraorbital Ridges						
Orbital Rims						
Posterior Zygomatic Arch						
Mastoid Processes						
Occipital Protuberance						
Mental Eminence						
Mandibular Ramus Flexure						

PELVIS	Male	?Male	?	?Female	Female	Unable
Sciatic Notch						
Preauricular Sulcus						
Subpubic Angle						
Ventral Arc						

Estimated Sex: _____

Notes: _____

Site:_____ Skel #:_____ Date:_____

ADULT AGE ESTIMATION

	L	R
Pubic Symphysis: Suchey-Brooks, 1990		
Auricular Surface: Lovejoy *et al.*, 1985		

Overall Estimated Age: _____

Dental Attrition: Brothwell, 1989	L M1	L M2	L M3	R M1	R M2	R M3
Maxilla						
Mandible						

Notes: _____

POSTCRANIAL MEASUREMENTS (MM) AND STATURE

	Left	Right
Femur		
Tibia		
Fibula		
Humerus		
Radius		
Ulna		

Estimated Stature: _____

(Stature: Trotter, 1970)

Notes: _____

Site:_____ Skel #:_____ Date:_____

SUBADULT AGE: FUSION

Element/Suture	Fused	Partially Fused	Unfused	Unobserv.	Notes
Metopic Suture					
Mental Symphysis					
Lateral to Basilar					
Lateral to Squamous					
Spheno-Occipital					
Hypoglossal Canals					
Cerv Neural Arch Midline					
Cerv Arch to Vert Body					
Thor Neural Arch Midline					
Thor Arch to Vert Body					
Lum Neural Arch Midline					
Lum Arch to Vert Body					
Annular Rings					
Scapula Coracoid					
Scapula Glen Cavity					
Scalupa Acromion					
Scapula Inferior Angle					
Scapula Med Border					
Clavicle Medial					
Humerus Greater Tub					
Humerus Head to Shaft					
Humerus Distal					
Humerus Med Epicondyle					
Radius Proximal					
Radius Distal					
Ulna Proximal					
Ulna Distal					
Ischium to Pubis					
Ischium to Ilium					
Ischial Epiphyses					
Iliac Crest					
Femur Head					
Greater Trochanter					
Lesser Trochanter					
Femur Distal					
Tibia Proximal					
Tibia Distal					
Fibula Proximal					
Fibula Distal					

Site:_____ Skel #:_____ Date:_____

SUBADULT AGE: FUSION (CONT'D)

Element/Suture	Fused	Partially Fused	Unfused	Unobserv.	Notes
S1-S2					
S2-S3					
S3-S4					
S4-S5					
Sacral Arches Midline					

Estimated Age From Fusion:

(Scheuer & Black, 2000)

Diaphyseal Length Age (Ubelaker, 1989)**:**

Notes:

SUBADULT AGE: DENTAL

		Right					Left				
Maxilla	Unable										
	Unerupted										
	Erupted										
Deciduous		m2	m1	c	i2	i1	i1	i2	c	m1	m2
Deciduous		m2	m1	c	i2	i1	i1	i2	c	m1	m2
Mandible	Unable										
	Unerupted										
	Erupted										

		Right								Left							
Maxilla	Unable																
	Unerupted																
	Erupted																
Permanent		M3	M2	M1	PM2	PM1	C	I2	I1	I1	I2	C	PM1	PM2	M1	M2	M3
Permanent		M3	M2	M1	PM2	PM1	C	I2	I1	I1	I2	C	PM1	PM2	M1	M2	M3
Mandible	Unable																
	Unerupted																
	Erupted																

Estimated Dental Age:

(Ubelaker, 1989)

Notes:

Site:_____ Skel #:_____ Date:_____

CRIBRA ORBITALIA

	Left	Right
Observable	Y / N	Y / N
CO Present	Y / N	Y / N
Type	Capillary	Capillary
	Porotic	Porotic
	Outgrowth	Outgrowth
	Other	Other
Description		

POROTIC HYPEROSTOSIS

Porotic Hyperostosis Present	Y / N / Unobs
Location	
Description	

PERIOSTEAL REACTION

Element		Left	Right
Tibia	Tibia Present	Y / N	Y / N
	Tibia Observable	Y / N	Y / N
	Periostitis Present	Y / N	Y / N
	Woven Bone	Y / N	Y / N
	Lamellar Bone	Y / N	Y / N
	Description		
Fibula	Fibula Present	Y / N	Y / N
	Fibula Observable	Y / N	Y / N
	Periostitis Present	Y / N	Y / N
	Woven Bone	Y / N	Y / N
	Lamellar Bone	Y / N	Y / N
	Description		

Site:_____ Skel #:_____ Date:_____

ADULT DENTAL ENAMEL HYPOPLASIA

		Right								Left							
Maxilla	Tooth Absent																
	Normal																
	Pit																
	Line																
	Groove																
		M3	M2	M1	PM2	PM1	C	I2	I1	I1	I2	C	PM1	PM2	M1	M2	M3
		M3	M2	M1	PM2	PM1	C	I2	I1	I1	I2	C	PM1	PM2	M1	M2	M3
Mandible	Tooth Absent																
	Normal																
	Pit																
	Line																
	Groove																

+=defect present

Notes:

SUBADULT DENTAL ENAMEL HYPOPLASIA

		Right					Left				
Maxilla	Tooth Absent										
	Normal										
	Pit										
	Line										
	Groove										
Deciduous		m2	m1	c	i2	i1	i1	i2	c	m1	m2
Deciduous		m2	m1	c	i2	i1	i1	i2	c	m1	m2
Mandible	Tooth Absent										
	Normal										
	Pit										
	Line										
	Groove										

+=defect present

Site:_____ Skel #:_____ Date:_____

CERVICAL

# Present		8th Present?	

Obs Clefting		Clefting Present	
Elements			

Transitional/Other

Obs Spond		Spond Present		
Elements		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B

Occipital Vert Present	Y / N	Occipitalization Present	Y / N
		Comp / Incomp / R / L	

THORACIC

# Present		13th Present?	

Obs Clefting		Clefting Present	
Elements			

Transitional/Other

Obs Spond		Spond Present		
Elements		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B

Site:_____ Skel #:_____ Date:_____

LUMBAR

# Present		6th Present?	

Obs Clefting		Clefting Present	
Elements			

Transitional/Other

Obs Spond		Spond Present		
Elements		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B

Sacralization	Element		Comp / Incomp / R / L
	Element		Comp / Incomp / R / L
Description			

SACRAL

# Present		6th Present?	

Obs Clefting		Clefting Present	
Elements			

Transitional/Other

Obs Spond		Spond Present		
Elements		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B
		R / L / B		R / L / B

Lumbarization	Element		Comp / Incomp / R / L
	Element		Comp / Incomp / R / L
Description			

Site:_____ Skel #:_____ Date:_____

OS ACROMIALE

Scapulae Observable	R L NO

Os Acromiale Present	R L NO
Separated Acrom Present	R L NO

RIB ANOMALIES

	Location	Uni/Bilateral	Description
		U / B	
		U / B	
		U / B	

Ribs Present	Y / N

Rib Anomalies Present	Y / N

Notes:

Site:_____ Skel #:_____ Date:_____

OTHER ANOMALIES

Anomaly	Element Affected	Side	Description
		R / L	
		R / L	
		R / L	
		R / L	

Observable Maxilla	R / L	Lip / Palate
Obs Hypoglossal Canals	R / L	Bipartite R / Bipartite L
Obs Distal Humerus	R / L	

Notes:

Site:_____ Skel #:_____ Date:_____

AGE:_____

AGE NOTES:_____

_____

_____

_____

BONES PRESENT:_____

_____

_____

_____

_____

OBSERVED ANOMALIES:_____

_____

_____

_____

_____

OTHER NOTES:_____

_____

_____

_____

_____

APPENDIX B
QUAKER BURIAL GROUND, COACH LANE, NORTH SHIELDS OSTEOLOGICAL DATA

List of Abbreviations for the North Shields Data:

CO cribra orbitalia
DEH dental enamel hypoplasia
DJD degenerative joint disease
F female
?F potential female
L left
M male
?M potential male
M1 first permanent molar
M2 second permanent molar
mos months
PH porotic hyperostosis
PM post-mortem
PM1 first permanent premolar
PNBF periosteal new bone formation
R right
U unable to determine sex
wiu weeks in utero
yrs years

Skeleton	Sex	Age	Stature	North Shields
COL10 002	-	34-38 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 003	-	Birth-1 yr	-	
	Stress Indicators -PH - alternative diagnosis is widespread infection			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	-	38-40 wiu	-	
	Stress Indicators			

004	none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10005	F	Adult	162.29cm ± 3.72	
	Stress Indicators -DEH			
	Congenital Defects -hypoplastic lamina - C1-L hypoplastic, R normal			

Skeleton	Sex	Age	Stature	North Shields
COL10006	-	1-1.5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10007	M	20-29 yrs	174.64cm ± 4.32	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	North Shields
COL10008	?M	40+ yrs	169.35cm ± 4.32	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R, L normal			
	Congenital Defects -hypoplastic lamina - C2-R hypoplastic, L normal, spinous process leans R, R inferior apophyseal facet displaced anteriorly & malformed, osteophyte on inferior body that articulates with osteophyte on C3 -hypoplastic lamina - C3-L hypoplastic, R normal, partial cleft on L lamina adjacent to spinous process, spinous process located			

	slightly to the R, R superior apophyseal facet displaced superiorly & flattened, osteophyte on superior body that articulates with osteophyte on C2

Skeleton	Sex	Age	Stature	North Shields
COL10 009	-	Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 010	?M	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 011	?F	Adult	150.85cm ± 4.30	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 012	?F	35-44 yrs	176.27cm ± 4.24	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 013	-	4-6 yrs	-	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - L5 -transitional vertebra thoracolumbar border - T11-both inferior			

	apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial

Skeleton	Sex	Age	Stature	North Shields
COL10 014	-	2-3 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 015	M	17-25 yrs	174.31cm ± 2.99	
	Stress Indicators -PNBF - tibia R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-inferior apophyseal facets face lateral; lumbarization T12-superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 016	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 017	?F	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects -hypoplastic lamina - L5-R, L normal, vertical measurements: R=15.7mm, L=21.5mm -hypoplastic lamina - S1-R, L normal, midline pushed to R side			

Skeleton	Sex	Age	Stature	North Shields
COL10 018	-	Birth-2 mos	-	
	Stress Indicators -CO - L, R normal			

	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 019	M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 020	-	~12 yrs	-	
	Stress Indicators -DEH -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 021	-	~3 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 022	F	20-29 yrs	unable	
	Stress Indicators none			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete on L, R missing PM -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	North Shields
COL10	-	Birth	-	
	Stress Indicators			

023	none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 024	-	~9 mos	-	
	Stress Indicators -CO - L, R normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 025	-	~4 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 026	?M	35-44 yrs	167.47cm ±4.05	
	Stress Indicators none			
	Congenital Defects -asymmetrical cranium - occipital sulcus veers R from the lambdoidal suture, internal & external occipital protuberances located to the R of centre -transitional vertebra lumbosacral border - sacralization L6-complete, R inferior apophyseal facet rudimentary, L unobservable -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	North Shields
COL10 027	?F	Adult	156.36cm ± 3.66	
	Stress Indicators none			
	Congenital Defects -hypoplastic lamina - C4-R, L normal, vertical measurements: R=6.0mm, L=8.7mm			

Skeleton	Sex	Age	Stature	North Shields
COL10028	-	Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10029	?F	Adult	161.12cm ± 4.45	
	Stress Indicators -CO - R & L -PNBF - tibia R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10030	M	20-29 yrs	164.23cm ± 3.27	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10031	-	30-32 wiu	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10032	M	Adult	173.01cm ± 4.05	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	-	Birth-2 mos	-	

033	**Stress Indicators** unobservable
	Congenital Defects none

Skeleton	Sex	Age	Stature	
COL10 034	F	40+ yrs	unable	North Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 035	?M	30-34 yrs	168.58cm ± 3.37	North Shields
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 036	?M	Adult	unable	North Shields
	Stress Indicators -PNBF - tibia L, R normal -PNBF - fibula R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 037	-	~Birth	-	North Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 038	M	20-25 yrs	178.08cm ± 2.99	North Shields
	Stress Indicators -CO - R & L			

	-DEH
	Congenital Defects -supernumerary vertebra - S6 -facet tropism - L5-R inferior apophyseal facet faces anterior -facet tropism - S1-R superior apophyseal facet faces posterior -os acromiale - R, L unobservable

Skeleton	Sex	Age	Stature	North Shields
COL10039	-	9 mos-1 yr	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10040	U	Adult	unable	
	Stress Indicators -PNBF - tibia R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10041	M	Adult	unable	
	Stress Indicators -CO - L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10042	M	Adult	174.63cm ± 3.37	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	M	Adult	172.75cm ± 4.32	
	Stress Indicators			

043	none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 044	M	30-39 yrs	161.13cm ± 3.27	
	Stress Indicators -CO - L, R normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 045	F	25-34 yrs	167.04cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 047	?F	Adult	unable	
	Stress Indicators -CO - R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 048	U	20-25 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 049	M	40-44 yrs	unable	
	Stress Indicators -PNBF - tibia R & L			
	Congenital Defects			

	-Klippel-Feil syndrome - C5-C6

Skeleton	Sex	Age	Stature	North Shields
COL10 050	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 051	?F	35-39 yrs	162.45cm ± 3.66	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R, L normal			
	Congenital Defects -transitional vertebra lumbosacral border - lumbarization T12-L superior apophyseal facet damaged PM but appears to face posteriorly, R missing PM, bilateral absent costal facets			

Skeleton	Sex	Age	Stature	North Shields
COL10 052	?F	25-29 yrs	158.96cm ± 4.30	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face laterally; lumbarization T12-L superior apophyseal facet faces medial, R missing PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 053	M	45+ yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 054	?M	Adult	167.47cm ± 4.05	
	Stress Indicators -DEH			

	-PNBF - tibia L, R normal
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 055	-	~1 yr	-	
	Stress Indicators -PH -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 056	?M	35-44 yrs	166.38cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-partial R, L normal -supernumerary vertebra - L6 -abnormal location of costal facets - T11-costal facets on transverse processes bilaterally -fused rib - possible L, fused starting about 3cm from heads, fused for about 5cm before edge of PM breakage, rest of ribs missing PM, radiograph show no signs of trauma			

Skeleton	Sex	Age	Stature	North Shields
COL10 057	-	~8 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - L5			

Skeleton	Sex	Age	Stature	North Shields
COL10 058	M	30-34 yrs	167.80cm ± 3.27	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior			

	apophyseal facet faces diagonally anterior to lateral & then wraps around to posterior edge, R missing PM; T12-both superior apophyseal facets face medial

Skeleton	Sex	Age	Stature	North Shields
COL10059	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10060	-	~5 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10061	?F	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10062	F	35-39 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10063	?F	25-29 yrs	163.98cm ± 3.55	
	Stress Indicators -PH - R parietal -CO - L, R normal -DEH			
	Congenital Defects			

	-transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6

Skeleton	Sex	Age	Stature	North Shields
COL10064	?F	30-39 yrs	159.08cm ± 3.72	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - C1 -hypoplastic lamina - C2-R, L normal -hypoplastic lamina - C3-R, L normal -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces diagonally anterior-lateral; lumbarization T12-R superior apophyseal facet faces medial, L missing PM, R costal facet rudimentary or even absent, L missing PM, rudimentary 12th R rib present -rudimentary apophyseal facet - C2-R inferior -rudimentary apophyseal facet - C3-R superior, R inferior -rudimentary apophyseal facet - C4-R superior, R inferior -rudimentary apophyseal facet - C5-R superior, R inferior -rudimentary apophyseal facet - C6-R superior			

Skeleton	Sex	Age	Stature	North Shields
COL10065	?F	30-39 yrs	162.04cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-L superior apophyseal facet faces medial, R faces posterior -broad rib - possibly bilateral 8th -broad rib - possible bilateral 9th			

Skeleton	Sex	Age	Stature	North Shields
COL10066	U	20-29 yrs	unable	
	Stress Indicators -DEH -PNBF - tibia R, L normal -PNBF - fibula R & L			

	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10067	M	Adult	173.03cm ± 3.27	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10068	-	~ 6 mos	-	
	Stress Indicators -PH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10069	-	9.5-10.5 yrs	-	
	Stress Indicators -DEH -PNBF - tibia R & L			
	Congenital Defects -transitional vertebra thoracolumbar vertebra - T11-L inferior apophyseal facet faces anterior, rest of vertebra too damaged PM for observation; lumbarization T12-R superior apophyseal facet faces medial, L faces posterior -rotated apophyseal facet - L3-inferior apophyseal facets face anterior -rotated apophyseal facet - L4-superior apophyseal facets face posterior, inferior apophyseal facets face partially anterior -rotated apophyseal facet - L5 superior apophyseal facets face partially posterior			

Skeleton	Sex	Age	Stature	North Shields
COL10070	?F	40+ yrs	157.03cm ± 3.55	
	Stress Indicators -CO - R, L normal -DEH			

	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10071	U	12+ yrs-Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10073	F	Adult	160.23cm ± 3.55	
	Stress Indicators -CO - R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10074	-	6 mos-1 yr	-	
	Stress Indicators -CO - L, R unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10075	-	3-4 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10076	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10077	?F	Adult	unable	
	Stress Indicators -CO - L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10078	?M	45+ yrs	166.77cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial -scoliosis - T1-R pedicle appears slightly shorter than L -scoliosis - T2-R pedicle appears shorter & thinner than L, R body thinner than L -scoliosis - T3-R pedicle extremely thin, L normal, superior body appears shifted L, inferior body appears shifted R, R body thinner than L -scoliosis - T4-L superior & inferior apophyseal facets smaller than R, R pedicle extremely thin, superior body appears shifted L, inferior body appears shifted R, R body thinner than L -scoliosis - T5-L superior & inferior apophyseal facets smaller than R, R pedicle thinner than L, superior body appears shifted L, costal facets not symmetrically placed -scoliosis - T6-L superior & inferior apophyseal facets smaller than R, L pedicle thinner than R, neural arch appears pulled to L -scoliosis -T7-L superior apophyseal facet smaller than R, L pedicle extremely thin, body appears pulled to R, L body about half as thick as R side, neural arch appears pulled to L -scoliosis - T8-L pedicle extremely thin, L body about half as thick as R side -scoliosis - T9-L pedicle extremely thin, L body about half as thick as R side -scoliosis - T10-L pedicle much shorter than R, inferior body possibly appears pulled to L but R side broken PM, bone of neural arch thinner on R -scoliosis - T11-L pedicle extremely short, inferior body appears pulled to L			

	-scoliosis - T12-L pedicle slightly shorter & thinner than R, R body thinner than L, neural arch appears pulled to R -scoliosis - L1-R body thinner than L; all other lumbar vertebrae too fragmented & damaged PM to observe -scoliosis - ribs-additional facets on ribs where they articulate with adjacent ribs -scoliosis - clavicle-R shortened with an exaggerated curve compared to L, radiograph shows no signs of trauma

Skeleton	Sex	Age	Stature	North Shields
COL10079	F	40-44 yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L missing PM; lumbarization T12-both superior apophyseal facets face medial, costal facets absent			

Skeleton	Sex	Age	Stature	North Shields
COL10080	?F	45+ yrs	156.34cm ± 3.55	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10081	-	Birth-2 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10082	?M	20-29 yrs	162.87cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 083	M	40-44 yrs	176.78cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - S3-S5 -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-superior apophyseal facets face medial -abnormal facet location - T11-additional? facet on superior edge of inferior apophyseal facet -bifid rib - possibly L, damaged PM but appears to split at sternal end for 6.5mm, fragment unusually broad & thin			

Skeleton	Sex	Age	Stature	North Shields
COL10 084	-	8-10 yrs	-	
	Stress Indicators -CO - L, R unobservable -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 085	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 086	?F	25-34 yrs	163.15cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces diagonally anterior-lateral, R faces anterior but is slightly inclined towards lateral; lumbarization T12-L superior apophyseal facets faces diagonally posterior-medial, R faces posterior but is slightly inclined towards medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 087	M	17-24 yrs	176.39cm ± 2.99	
	Stress Indicators -CO- R & L -DEH			
	Congenital Defects -transitional vertebra cervicothoracic border - cervical ribs C7- bilateral possibly, R=3.3mm long, L broken PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 088	?M	25-35 yrs	unable	
	Stress Indicators -CO - R, L unobservable -PNBF - tibia R & L -PNBF - fibula R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 089	?M	25-35 yrs	170.02cm ± 2.99	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 090	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects -supernumerary vertebra - L6 -rudimentary apophyseal facet - L6-R inferior -rudimentary apophyseal facet - S1-R superior			

Skeleton	Sex	Age	Stature	North Shields
COL10	-	~6 mos	-	
	Stress Indicators			

091	none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10092	?F	20-29 yrs	163.52cm ± 3.72	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-L superior apophyseal facet faces medial, R faces posterior, L costal facet is convex, R costal facet is flat, rudimentary L 12th rib -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	North Shields
COL10093	M	45+ yrs	167.08cm ± 3.27	
	Stress Indicators -DEH			
	Congenital Defects -spondylolysis - L4-bilateral -spondylolysis - L5-bilateral?, L damaged PM but appears to be affected			

Skeleton	Sex	Age	Stature	North Shields
COL10094	?F	30-39 yrs	154.94cm ± 4.24	
	Stress Indicators -CO - R, L unobservable -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces both anterior & lateral, L faces lateral; lumbarization T12-R superior apophyseal facet faces both posterior & medial, L faces medial -supernumerary vertebra - S6 -os acromiale - R & L			

Skeleton	Sex	Age	Stature	North Shields
COL10	-	1.5-2 yrs	-	

095	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10096	M	30-34 yrs	172.08cm ± 3.27	
	Stress Indicators -DEH -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	North Shields
COL10097	?M	35-44 yrs	175.61cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10098	?F	20-29 yrs	154.00cm ± 4.24	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10099	?F	30-34 yrs	163.71cm ± 3.55	
	Stress Indicators -CO - R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior with a slight slope towards lateral; lumbarization T12-L superior apophyseal facet faces medial, R faces diagonal posterior-medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 100	-	6-9 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 101	?F	25-34 yrs	161.11cm ± 4.24	
	Stress Indicators -PNBF - tibia L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 102	?M	Adult	167.32cm ± 3.37	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - C7 -aplastic lamina - C7-R -hypoplastic lamina - C6-L, R normal & about twice the size of L, aplastic R inferior apophyseal facet			

Skeleton	Sex	Age	Stature	North Shields
COL10 103	?F	25-34 yrs	148.83cm ± 3.55	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 104	-	2-3 yrs	-	
	Stress Indicators -CO - R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 105	-	12-18 mos	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 107	-	15-20 yrs	-	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - L5 -spondylolysis - L5-bilateral			

Skeleton	Sex	Age	Stature	North Shields
COL10 108	-	~18 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 109	M	25-29 yrs	168.07cm ± 2.99	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 110	-	10-12 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 111	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 112	?M	30-34 yrs	171.45cm ± 2.99	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects -os acromiale - L, R normal			

Skeleton	Sex	Age	Stature	North Shields
COL10 113	?F	30-34 yrs	160.37cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -hypoplastic lamina - C5-R & L, height measurements: R=2.7mm, L=5.7mm, for comparison C4 height measurements: R=9.4mm, L=10.0mm			

Skeleton	Sex	Age	Stature	North Shields
COL10 114	U	40-44 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 115	M	25-34 yrs	169.50cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -spondylolysis - L5-L, R normal -rudimentary costal facet - T12-R costal facet flat, L costal facet ~1/3 size of R			

	-rudimentary rib - L 12th rib-measurements: R=78.4mm, L=37.7mm, R head flat, L head concave -os acromiale - R, L normal

Skeleton	Sex	Age	Stature	North Shields
COL10 116	?F	Adult	unable	
	Stress Indicators -PNBF - tibia R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 117	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 118	-	~2 yrs	-	
	Stress Indicators -CO - L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 119	M	40-44 yrs	157.15cm ± 2.99	
	Stress Indicators -PNBF - tibia L, R normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-both superior apophyseal facets face posterior, both inferior apophyseal facets face anterior; lumbar ribs L1-both superior apophyseal facets face posterior, R inferior apophyseal facet faces anterior, L faces lateral, costal facets present R & L located on superior apophyseal facets; L2-R superior apophyseal facet faces posterior, L faces medial -abnormal location of costal facets - T12- R & L facets located where transverse processes should have been in addition to costal facets on body			

	-abnormal location of costal facets - L1-R & L located on superior apophyseal facets

Skeleton	Sex	Age	Stature	North Shields
COL10 120	?M	40-44 yrs	170.54cm ± 2.99	
	Stress Indicators -DEH -PNBF - tibia L, R normal -PNBF - fibula R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 121	?M	30-39 yrs	170.28cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 122	-	12-16 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral although damaged PM, R faces anterior; lumbarization T12-L superior apophyseal facet faces medial, R faces posterior			

Skeleton	Sex	Age	Stature	North Shields
COL10 123	M	30-34 yrs	166.06cm ± 3.37	
	Stress Indicators -DEH			
	Congenital Defects -rotated apophyseal facet - L5-both inferior apophyseal facets face anterior -rotated apophyseal facet - S1-both superior apophyseal facets face posterior			

Skeleton	Sex	Age	Stature	North Shields
COL10 124	-	2-3 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 125	M	45+ yrs	165.04cm ± 3.29	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - C1 -transitional vertebra occipitocervical border - occipitalization C1- incomplete R & L, not fused anterior or R posterior, facet for dens appears almost rudimentary, C2 missing PM -transitional vertebra cervicothoracic border - cervical rib C7- possibly bilateral, costal facet on L transverse process, R missing PM, possible costal facet on R superior body, L missing PM, possible costal facets on inferior body R & L			

Skeleton	Sex	Age	Stature	North Shields
COL10 126	?M	25-34 yrs	167.72cm ± 3.29	
	Stress Indicators -DEH -PNBF - tibia L, R normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-L inferior apophyseal facet faces anterior, R faces lateral; L1-L superior apophyseal facet faces posterior, L faces medial, R transverse process looks like T12 process, L transverse process looks like normal lumbar process			

Skeleton	Sex	Age	Stature	North Shields
COL10 127	-	15-18 yrs	-	
	Stress Indicators -CO - L, R normal -DEH -PNBF - tibia R & L -PNBF - fibula R & L			

	Congenital Defects -supernumerary vertebra - L6

Skeleton	Sex	Age	Stature	North Shields
COL10 128	M	30-34 yrs	170.86cm ± 4.05	
	Stress Indicators -DEH			
	Congenital Defects -Klippel-Feil syndrome - C6-C7			

Skeleton	Sex	Age	Stature	North Shields
COL10 129	?F	25-34 yrs	unable	
	Stress Indicators -PNBF - tibia R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 130	?F	30-34 yrs	156.06cm ± 3.55	
	Stress Indicators -DEH -PNBF - tibia R, L normal -PNBF - fibula R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L missing PM; lumbarization T12-R superior apophyseal facets faces medial, L missing PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 131	-	~1 yr	-	
	Stress Indicators -PH -CO - L, R normal -DEH			
	Congenital Defects -broad rib - R, sternal ⅓ to ¼ of rib broadened			

Skeleton	Sex	Age	Stature	North Shields
COL10	U	Adult	unable	

132	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 133	?F	Adult	unable	
	Stress Indicators -CO - L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 134	?M	25-34 yrs	173.14cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L faces posterior -abnormal location of costal facets - T12-R & L costal facets on transverse processes instead of body			

Skeleton	Sex	Age	Stature	North Shields
COL10 135	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 136	-	12-15 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	?M	30-34 yrs	173.66cm ± 2.99	

137	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 138	M	35+ yrs	174.38cm ± 3.37	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 139	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 140	M	25-34 yrs	161.57cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L5-imcomplete R -rudimentary apophyseal facet - L5-both inferior apophyseal facets -rudimentary apophyseal facet - S1-both superior apophyseal facets			

Skeleton	Sex	Age	Stature	North Shields
COL10 141	U	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects -asymmetrical cranium - external occipital protuberance skewed to R & bulges further out on R side than L, internal occipital sulcus curves to L, R cerebral fossa much larger than L, area below internal occipital protuberance missing PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 142	?F	25-29 yrs	unable	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R missing PM; lumbarization T12-L superior apophyseal facet faces medial, R missing PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 143	?F	35-39 yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 144	-	4.5-7.5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 145	?M	30-34 yrs	161.96cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -Klippel-Feil syndrome - C2-C3			

Skeleton	Sex	Age	Stature	North Shields
COL10 146	?F	Adult	174.13cm ± 3.55	
	Stress Indicators -PNBF - tibia R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 147	-	~9 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 148	-	15-18 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects -cleft neural arch - S1-S4			

Skeleton	Sex	Age	Stature	North Shields
COL10 150	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 151	?M	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face diagonally anterior-lateral; lumbarization T12-L superior apophyseal facet faces medial, R missing PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 152	-	~7 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 153	-	3-5 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 154	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 155	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 156	-	~3 yrs	-	
	Stress Indicators none			
	Congenital Defects -bifid rib - unknown location, 2 sternal facets present			

Skeleton	Sex	Age	Stature	North Shields
COL10 157	-	~12 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-both inferior apophyseal facets face anterior; L1-both superior apophyseal facets face posterior, no costal facets present			

Skeleton	Sex	Age	Stature	North Shields
COL10	?M	Adult	168.26cm ± 3.29	

158	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	
COL10 159	-	9-10 yrs	-	North Shields
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-both inferior apophyseal facets face anterior; L1-both superior apophyseal facets face posterior, no costal facets present			

Skeleton	Sex	Age	Stature	
COL10 160	-	~ 6 mos	-	North Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 161	?M	35-44 yrs	unable	North Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 162	M	45+ yrs	170.41cm ± 2.99	North Shields
	Stress Indicators none			
	Congenital Defects -cleft neural arch - S1-S2			

Skeleton	Sex	Age	Stature	
COL10	?M	Adult	unable	North Shields
	Stress Indicators			

163	none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 164	F	30-39 yrs	152.31cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - C1 -scoliosis - C5-R superior apophyseal facet half the size of L, L inferior apophyseal facet half the size of L, L pedicle thinner than R, R lamina curves in, L is normal -scoliosis - C6- R superior apophyseal facet half the size of L, L inferior apophyseal facet half the size of L, L pedicle thinner than R, R lamina curves in, L is normal, body flattened on L side -scoliosis - C7- R superior apophyseal facet half the size of L, L inferior apophyseal facet half the size of L, L pedicle thinner than R -scoliosis - T1-body flatter on R, DJD R inferior apophyseal facet -scoliosis - T2-DJD R superior & inferior apophyseal facets -scoliosis - T3-R pedicle extremely thin, DJD R superior & inferior apophyseal facets -scoliosis - T4- R pedicle extremely thin, DJD R superior & inferior apophyseal facets -scoliosis - T5- R pedicle extremely thin, DJD R superior & inferior apophyseal facets -scoliosis - T6- R pedicle extremely thin, DJD R superior apophyseal facets -scoliosis - T7-appears normal -scoliosis - T8-L superior apophyseal facet half the size of R, L pedicle may be thinner than R (damaged PM), DJD L inferior apophyseal facet -scoliosis - T9-pedicles damaged PM, DJD L superior & inferior apophyseal facets, fused to T10 on L side of neural arch (body missing PM) -scoliosis - T10-pedicles about even in size, DJD superior & inferior apophyseal facets, fused to T9 on L side (body missing PM) -scoliosis - T11-pedicles damaged PM, DJD L superior & inferior apophyseal facets -scoliosis - T12-pedicles even, DJD L superior apophyseal facet -scoliosis - unknown thoracic body-compressed on R, lower half of			

	body skewed to R -scoliosis - L1-normal -scoliosis - L2-lower half of neural arch skewed towards R -scoliosis - L3-lower half of neural arch skewed towards L -scoliosis - ~C6-T6-curves to R -scoliosis - T7-T11-curves to L

Skeleton	Sex	Age	Stature	North Shields
COL10 165	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 166	-	6-18 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 167	F	38-44 yrs	157.31cm ± 4.24	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 168	M	35-39 yrs	170.80cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -supernumerary vertebra - S6			

Skeleton	Sex	Age	Stature	North Shields
COL10	?M	20-24 yrs	169.84cm ± 3.37	
	Stress Indicators			

170	-CO - R & L -DEH
	Congenital Defects -transitional vertebra cervicothoracic border - cervical rib C7-R, measurement: R=15.6mm, L unobservable -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-R superior apophyseal facet faces medial, L missing PM, both costal facets rudimentary, L rudimentary rib & R rudimentary rib head present

Skeleton	Sex	Age	Stature	North Shields
COL10 171	U	35+ yrs	unable	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces diagonally anterior-lateral, R missing PM; lumbarization T12-L superior apophyseal facet faces medial, R faces diagonally posterior-medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 172	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 174	M	30-39 yrs	172.36cm ± 3.37	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 175	?M	17-20 yrs	174.31cm ± 2.99	
	Stress Indicators -PNBF - tibia L, R normal -PNBF - fibula L, R normal			
	Congenital Defects			

	none

Skeleton	Sex	Age	Stature	North Shields
COL10 176	M	35-39 yrs	166.64cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - lumbarization S1-complete, both inferior apophyseal facets are rudimentary			

Skeleton	Sex	Age	Stature	North Shields
COL10 177	?F	35-44 yrs	177.70cm ± 4.24	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medially			

Skeleton	Sex	Age	Stature	North Shields
COL10 178	?M	Adult	162.63cm ± 3.29	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 179	-	3-4 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 181	U	40-44 yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-neural arch			

	missing PM; lumbarization T12-R superior apophyseal faces medial, L faces posterior

Skeleton	Sex	Age	Stature	North Shields
COL10 183	-	2-3 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 186	-	3-4 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 191C	M	Adult	160.18cm ± 3.27	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects -asymmetrical cranium - R parietal eminence lower than L, R occipitomastoid suture not very visible especially when compared to L side, frontal does not line up with occipital but appears to be due to deformation PM, greater wear on mandibular R M1, R M2, & R PM1 compared to L M1: maybe due to asymmetry?, maxilla missing PM, L temporomandibular articular surface nearly double the size of R, L mandibular condyle bigger anterior-posteriorly than R			

Skeleton	Sex	Age	Stature	North Shields
COL10 193C	F	45+ yrs	unable	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior			

	apophyseal facets face medial, both costal facets rudimentary

Skeleton	Sex	Age	Stature	North Shields
COL10 195C	U	17-25 yrs	unable	
	Stress Indicators -CO - L, R unobservable -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 196C	M	Adult	168.27cm ± 3.27	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 197C	-	2-4 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 199AC	M	30-34 yrs	172.38cm ± 4.32	
	Stress Indicators none			
	Congenital Defects -facet tropism - L4-L inferior apophyseal facet faces anterior -facet tropism - L5-L superior apophyseal facet faces posterior			

Skeleton	Sex	Age	Stature	North Shields
COL10 199SC	-	4-7 yrs	-	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - L5-just L of centre			

Skeleton	Sex	Age	Stature	North Shields
COL10 200C	?M	Adult	178.87cm ± 4.05	
	Stress Indicators -CO - R, L normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 205	-	2-3 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 206	?M	17-25 yrs	162.61cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 207	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 208	-	~2 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	-	Birth-5 mos	-	

209	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 210	M	25-34 yrs	178.34cm ± 2.99	
	Stress Indicators -PNBF - fibula R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facets face lateral, L faces anterior; lumbarization T12-R superior apophyseal facets faces medial, L faces posterior, costal facets asymmetrical: R located at top of pedicle, L at bottom -facet tropism - L1-L inferior apophyseal facet faces anterior, R faces lateral -facet tropism - L2-L superior apophyseal facet faces posterior, R faces medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 211	?F	Adult	150.85cm ± 4.30	
	Stress Indicators none			
	Congenital Defects -elongated styloid process - L measures 34.5mm, end broken off PM, angled anteriorly & laterally, slightly wavy in contour, R unobservable (broken off PM)			

Skeleton	Sex	Age	Stature	North Shields
COL10 212	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 213	M	Adult	unable	
	Stress Indicators none			
	Congenital Defects			

	-fused rib - possibly lower L, bony bridge just lateral to the head, about 16mm wide, heads & rest of ribs broken & missing PM

Skeleton	Sex	Age	Stature	North Shields
COL10 214	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 215	-	1.5-2 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 216	M	35-39 yrs	175.79cm ± 4.05	
	Stress Indicators -CO - R & L -DEH -PNBF - fibula L, R normal			
	Congenital Defects -transitional vertebra cervicothoracic border - cervical rib C7- possibly, R side, measures 17mm & broken PM, L side broken PM -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 217	?F	20-24 yrs	168.83cm ± 3.66	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 218	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 219	?M	20-29 yrs	177.33cm ± 4.05	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	North Shields
COL10 220	?F	Adult	unable	
	Stress Indicators -PNBF - tibia R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 221	?F	25-34 yrs	161.11cm ± 4.24	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 222	-	~2 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	?F	Adult	158.58cm ± 3.72	

223	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	
COL10 224	-	~2 yrs	-	North Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 225	?F	40-44 yrs	unable	North Shields
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L missing PM; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	
COL10 226	F	Adult	154.81cm ± 3.55	North Shields
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 227	-	~3 yrs	-	North Shields
	Stress Indicators -CO - R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10 228	-	36-40 wiu	-	North Shields
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 229	U	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 230	-	~12 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 231	-	~6 mos	-	
	Stress Indicators -PH -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 232	M	35-39 yrs	174.57cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 233	F	25-34 yrs	157.31cm ± 3.55	
	Stress Indicators none			
	Congenital Defects			

	-transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-both superior apophyseal facets missing PM

Skeleton	Sex	Age	Stature	North Shields
COL10 234	?F	35-39 yrs	167.32cm ± 3.55	
	Stress Indicators -CO - R, L normal -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 235	-	Birth	-	
	Stress Indicators unobservable			
	Congenital Defects -block vertebrae - two unknown thoracic vertebrae-fused on R side of neural arch, pedicles & transverse processes unfused, spinous process missing PM, unfused to bodies, bodies missing PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 236	F	Adult	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 237	-	6-8 yrs	-	
	Stress Indicators -CO - L, R unobservable -PNBF - tibia R & L -PNBF - fibula L, R unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 238	-	~1 yr	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 239	-	36-38 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 240	-	5-9 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 242	M	35+ yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 243	M	30-39 yrs	179.17cm ± 3.37	
	Stress Indicators -DEH -PNBF - tibia R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L missing PM			

Skeleton	Sex	Age	Stature	North Shields
COL10 244	-	36-38 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 245	-	1-3 yrs	-	
	Stress Indicators -CO - R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 246	-	~2 yrs	-	
	Stress Indicators -DEH -PNBF - fibula L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 247	M	20-29 yrs	173.32cm ± 4.05	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 248	-	~5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	?M	Adult	unable	

249	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10 250	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 251	F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 252	-	14-16 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - L6 -transitional vertebra lumbosacral border - sacralization L6-still in process of fusing due to individual's age so cannot tell if complete or not -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	North Shields
COL10 253	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10	?F	Adult?	unable	

254	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	
COL10255	?M	Adult	unable	North Shields
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10256	-	~Birth	-	North Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10257	M	45+ yrs	182.31cm ± 3.27	North Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10258	?F	35-44 yrs	159.32cm ± 3.72	North Shields
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
COL10259	M	35-39 yrs	170.60cm ± 3.37	North Shields
	Stress Indicators none			
	Congenital Defects			

	-transitional vertebra thoracolumbar border - T12-both inferior apophyseal facets missing PM; L1-R superior apophyseal facet faces posterior, L missing PM, no costal facets present

Skeleton	Sex	Age	Stature	North Shields
COL10 260	-	1.5-2 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 261	?F	35-39 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 262	F	45+ yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10 263	M	45+ yrs	181.12cm ± 3.27	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-L superior apophyseal facet faces medial, R faces posterior			

Skeleton	Sex	Age	Stature	North Shields
COL10 264	M	20-29 yrs	174.70cm ± 2.99	
	Stress Indicators -DEH			

	Congenital Defects none

Skeleton	Sex	Age	Stature	North Shields
COL10265	M	25-34 yrs	171.32cm ± 2.99	
	Stress Indicators -DEH -PNBF - fibula L, R normal			
	Congenital Defects -cleft neural arch - L6 -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	North Shields
COL10267	?F	40+ yrs	169.12cm ± 3.66	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	North Shields
COL10268C	M	20-29 yrs	unable	
	Stress Indicators -CO - R & L			
	Congenital Defects none			

APPENDIX C
ST HILDA'S CHURCH, CORONATION STREET, SOUTH SHIELDS OSTEOLOGICAL DATA

List of Abbreviations for the South Shields Data:

CO	cribra orbitalia	mos	months
DEH	dental enamel hypoplasia	PH	porotic hyperostosis
DJD	degenerative joint disease	PM	post-mortem
F	female	PNBF	periosteal new bone formation
?F	potential female	R	right
L	left	U	unable to determine sex
M	male	wiu	weeks in utero
?M	potential male	yrs	years

Skeleton	Sex	Age	Stature	South Shields
CS06 51	M	45+ yrs	180.94cm ± 2.99	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial, possibly a rudimentary convex costal facet present on L side but unwashed so difficult to tell, R side of body missing PM -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	South Shields
CS06 62	M	35-39 yrs	170.93cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - lumbar ribs L1-costal			

	facet R side with normal appearance, L side costal facet flat & long, no obvious lumbar ribs or ribs with unusual head morphology located -transitional vertebra lumbosacral border - sacralization L6-incomplete L, fused at L transverse process & L neural arch, body unfused, R transverse process articulates with S1 ala, fused twisted to L, spinous process to L of centre -supernumerary vertebra - L6 -aplastic ulnar styloid process - L, R is normal

Skeleton	Sex	Age	Stature	South Shields
CS06 70	M	30-34 yrs	170.15cm ± 2.99	
	Stress Indicators -DEH -PNBF - tibia L, R normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	South Shields
CS06 75	?F	Adult	155.92cm ± 3.55	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 80a	F	20-29 yrs	160.37cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 80b	-	32-34 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 89	-	1-2 yrs	-	
	Stress Indicators -CO - L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 96	-	7-10 yrs	-	
	Stress Indicators -CO - R & L			
	Congenital Defects -cleft neural arch - S3 -transitional vertebra thoracolumbar border - T12-both inferior apophyseal facets face anterior; L1-both superior apophyseal facets face posterior, no costal facets present			

Skeleton	Sex	Age	Stature	South Shields
CS06 100	M	35-39 yrs	171.32cm ± 2.99	
	Stress Indicators -PNBF - tibia R & L			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L5-complete but not fused at apophyseal facets -elongated styloid process - R & L, measurements: R=33.5mm but broken at end PM, L=46.5mm, R is angled anteriorly, L is angled anteriorly & medially, L smooth aside from one bulge			

Skeleton	Sex	Age	Stature	South Shields
CS06 107	F	45+ yrs	160.37cm ± 3.55	
	Stress Indicators -DEH -PNBF - tibia R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-both inferior apophyseal facets face lateral; lumbarization T13-both superior apophyseal facets face medial, both costal facets convex & small, no unusual ribs located -supernumerary vertebra -T13 -supernumerary ribs - costal facets on both sides of T13, no			

	associated ribs located -pectus carinatum -os acromiale - R & L

Skeleton	Sex	Age	Stature	South Shields
CS06 111	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 116	-	1-2 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 122	U	30-34 yrs	unable	
	Stress Indicators -DEH -PNBF - tibia R & L -PNBF - fibula L, R unobservable			
	Congenital Defects -cleft neural arch - L6 -transitional vertebra lumbosacral border - sacralization L6- incomplete L, fused at L transverse process, unfused at body, apophyseal facets, & R transverse process, S1 compressed on L side perhaps to accommodate sacralization -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 124	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 125	M	40+ yrs	175.48cm ± 2.99	
	Stress Indicators -PNBF - tibia L, R normal -PNBF - fibula L, R normal			
	Congenital Defects -spondylolysis - L5-bilateral			

Skeleton	Sex	Age	Stature	South Shields
CS06 127	F	40-44 yrs	150.64cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	South Shields
CS06 131	-	Birth-6 mos	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 135	M	45+ yrs	175.48cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - lumbar rib L1-costal facet instead of transverse process on R side, no lumbar rib recovered, apophyseal facets & L transverse process normal			

Skeleton	Sex	Age	Stature	South Shields
CS06 140	-	15-17 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 145	-	3-4 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 149	-	36-40 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 154	-	1-3 yrs	-	
	Stress Indicators -CO - R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 159	F	45+ yrs	162.73cm ± 3.55	
	Stress Indicators -DEH -PNBF - tibia L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 165	-	3-4 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06	-	~2 yrs	-	

170	**Stress Indicators** -CO - R & L
	Congenital Defects none

Skeleton	Sex	Age	Stature	South Shields
CS06 174	-	10-12 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 179	?F	35-39 yrs	161.20cm ± 3.55	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 184	?F	Adult	162.53cm ± 3.72	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 189	?F	40-44 yrs	159.08cm ± 3.72	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 195	U	30-34 yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects			

	none

Skeleton	Sex	Age	Stature	South Shields
CS06 200	?F	35-44 yrs	163.43cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - lumbarization T12-absent R costal facet, L facet normal but located on pedicle, apophyseal facets normal			

Skeleton	Sex	Age	Stature	South Shields
CS06 203	F	30-39 yrs	159.12cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial -aplastic apophyseal facet - L5-L inferior apophyseal facet, area of neural arch where it should have been has porous surface similar to that seen in os acromiale or spondylolysis, no separate apophyseal facet located, S1 L apophyseal facet normal but slightly smaller than R, DJD L5 & S1 R apophyseal facets			

Skeleton	Sex	Age	Stature	South Shields
CS06 208	M	45+ yrs	179.77cm ± 2.99	
	Stress Indicators -DEH -PNBF - tibia R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 214	-	1.5-3 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 217	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 222	F	20-29 yrs	158.01cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 228	-	15-17 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - S3-S5 -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	South Shields
CS06 235	M	45+ yrs	171.06cm ± 2.99	
	Stress Indicators -PNBF - tibia L, R normal -PNBF - fibula L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 238.1	?F	45+ yrs	153.96cm ± 3.55	
	Stress Indicators -PNBF tibia R, L normal			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L5-complete but L transverse process is not fused			

Skeleton	Sex	Age	Stature	South Shields
CS06 238.ii	-	~4 yrs	-	
	Stress Indicators -CO - R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 241	F	Adult	16315cm ± 3.55	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 243	?M	30-34 yrs	168.75cm ± 3.27	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal missing PM, L faces posterior			

Skeleton	Sex	Age	Stature	South Shields
CS06 248	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 249	U	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06	-	~Birth	-	

254	**Stress Indicators** none
	Congenital Defects none

Skeleton	Sex	Age	Stature	South Shields
CS06 256	?M	35-39 yrs	164.30cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 264	?M	45+ yrs	169.59cm ± 3.37	
	Stress Indicators -CO - R & L -PNBF - tibia R, L normal			
	Congenital Defects -transitional vertebra cervicothoracic border - cervical rib C7-R, measurements: 14.6mm thick, 20.0mm long, angles anteriorly & inferiorly, L side missing PM -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	South Shields
CS06 268	?M	35-39 yrs	167.55cm ± 2.99	
	Stress Indicators -DEH -PNBF - tibia L, R normal -PNBF - fibula L, R unobservable			
	Congenital Defects -transitional vertebra lumbosacral border - L6-complete -supernumerary vertebra L6 -os acromiale - R, L unobservable			

Skeleton	Sex	Age	Stature	South Shields
CS06	F	40+ yrs	161.48cm ± 3.55	

274	**Stress Indicators** -DEH
	Congenital Defects -asymmetrical cranium - L lambdoidal suture lower on R making L parietal appear larger than R, R & L lambdoidal sutures partially obliterated but likely due to age, L parietal boss lower than R, L side of occipital protrudes further than R side -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L faces posterior -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6

Skeleton	Sex	Age	Stature	
CS06 284	U	45+ yrs	unable	South Shields
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
CS06 289	M	40-44 yrs	177.69 cm ± 2.99	South Shields
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
CS06 294	U	Adult	unable	South Shields
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	
CS06 300	F	30-34 yrs	158.42cm ± 3.55	South Shields
	Stress Indicators -DEH -PNBF - tibia R & L			

	-PNBF - fibula R & L
	Congenital Defects -cleft neural arch - S1 & S3-S5

Skeleton	Sex	Age	Stature	South Shields
CS06 302	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 305	M	30-34 yrs	179.67cm ± 3.37	
	Stress Indicators -DEH -PNBF - tibia R, L normal -PNBF - fibula, R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 309	F	30-34 yrs	167.27cm ± 4.24	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 311	-	36-40 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 315	M	Adult	169.63cm ± 4.05	
	Stress Indicators -CO - R, L normal -DEH			

	-PNBF - tibia R & L -PNBF - fibula L, R normal
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-L superior apophyseal facet faces medial, R faces posterior -scoliosis - T2-DJD R inferior apophyseal facet -scoliosis - T3-R inferior apophyseal facet fused to T4, pedicles normal, body compressed on R side, DJD R superior apophyseal facet -scoliosis - T4-R superior apophyseal facet fused to T3, L inferior apophyseal facet & R neural arch fused to T5, pedicles normal, body compressed on R side -scoliosis - T5-L superior apophyseal facet & R neural arch fused to T4, L pedicle extremely thin, body compressed on L side, DJD R inferior apophyseal facet -scoliosis - T6-L inferior apophyseal facet fused to T7, L pedicle extremely thin, body greatly compressed on L side & fused to T7 -scoliosis - T7-L superior apophyseal facet fused to T6, L pedicle very thin, body greatly compressed on L side & fused to T6, DJD L inferior apophyseal facet -scoliosis - T1-T2, T8-T12-no changes seen from scoliosis -scoliosis - ribs-fragmentary but no changes from scoliosis seen

Skeleton	Sex	Age	Stature	South Shields
CS06 320	-	3.5-4.5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 321	M	40+ yrs	180.89cm ± 3.27	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06	M	35-39 yrs	164.56cm ± 2.99	
	Stress Indicators			

323	-DEH
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L faces posterior -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6

Skeleton	Sex	Age	Stature	South Shields
CS06 332	M	30-39 yrs	168.07cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -elongated styloid process - R measures 53.3mm, points posterior, L missing PM			

Skeleton	Sex	Age	Stature	South Shields
CS06 338	-	3-4 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 344	F	20-29 yrs	165.65cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - L5 -cleft neural arch - S3-S5 -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	South Shields
CS06 348	M	Adult	175.74cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 353	M	25-29 yrs	181.46cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-R inferior apophyseal facet faces anterior, L faces lateral; L1-R superior apophyseal facet faces posterior, L faces medial, no costal facets present -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 358	M	Adult	168.39cm ± 4.05	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 362	-	36-38 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 365	-	32-36 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 371	M	30-39 yrs	182.50cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - L6			

	-spondylolysis - L5-bilateral -transitional vertebra lumbosacral border - sacralization L6-complete L side, R side too damaged PM to observe -supernumerary vertebra lumbosacral border - L6

Skeleton	Sex	Age	Stature	South Shields
CS06 375	U	40-44 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 377	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 381	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 385	-	~2 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 390	M	40-44 yrs	174.88cm ± 3.37	
	Stress Indicators -DEH			
	Congenital Defects -spondylolysis - L5-L, R unobservable			

	-block vertebrae - L-possibly L3-L4, only R side of neural arch & portion of bodies present: all fused -os acromiale - L, R unobservable

Skeleton	Sex	Age	Stature	South Shields
CS06 398	-	1-3 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 401	-	Birth-5 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 404	?F	45+ yrs	154.54cm ± 3.57	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial, costal facets not present -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	South Shields
CS06 410	?M	20-24 yrs	168.85 cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 419	U	45+ yrs	unable	
	Stress Indicators -PNBF - tibia L, R normal			

	-PNBF - fibula R & L
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial

Skeleton	Sex	Age	Stature	South Shields
CS06 430	M	30-34 yrs	172.56cm ± 3.27	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - T1 -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial -aplastic transverse process - L1-R			

Skeleton	Sex	Age	Stature	South Shields
CS06 435	M	25-29 yrs	179.92cm ± 3.37	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 440	F	20-29 yrs	165.93cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -hypoplastic lamina - S1-L, R normal, results in normal cleft neural arch			

Skeleton	Sex	Age	Stature	South Shields
CS06 442	M	45+ yrs	176.39cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial, no costal facet L side, R side has			

	knob where transverse process should be: maybe articulation for rib?, no unusual ribs located

Skeleton	Sex	Age	Stature	South Shields
CS06 447	?M	40-44 yrs	173.27cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -facet tropism - L5-L inferior apophyseal facet faces anterior, R faces lateral -facet tropism - S1-L superior apophyseal facet faces posterior, R faces medial -os acromiale - R, L normal			

Skeleton	Sex	Age	Stature	South Shields
CS06 453	-	~3 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 467	-	28-30 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 468	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 472	U	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects			

	none

Skeleton	Sex	Age	Stature	South Shields
CS06 479	F	35-39 yrs	153.70cm ± 3.55	
	Stress Indicators -DEH -PNBF - tibia R & L			
	Congenital Defects -transitional vertebra lumbosacral border - lumbarization S1-incomplete, fused to S2 at R ala, unfused at body, neural arch, apophyseal facets, & most of L ala			

Skeleton	Sex	Age	Stature	South Shields
CS06 492	M	30-34 yrs	176.14cm ± 3.37	
	Stress Indicators -DEH			
	Congenital Defects -spondylolysis - L5-R, L normal -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-R superior apophyseal facet faces medial, L missing PM, costal facets a little small			

Skeleton	Sex	Age	Stature	South Shields
CS06 497	?M	40-44 yrs	170.80cm ± 2.99	
	Stress Indicators -PNBF - tibia R, L normal -PNBF - fibula R, L unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 502	F	45+ yrs	164.82cm ± 3.55	
	Stress Indicators -DEH -PNBF - tibia L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 507	?F	45+ yrs	169.99cm ± 3.66	
	Stress Indicators -PNBF - tibia L, R unobservable			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 512	M	30-39 yrs	165.18cm ± 3.27	
	Stress Indicators -DEH -PNBF - fibula R & L			
	Congenital Defects -spondylolysis - L5-bilateral			

Skeleton	Sex	Age	Stature	South Shields
CS06 517	?F	35-39 yrs	161.20cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 519	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 523	?M	35-39 yrs	166.37cm ± 3.27	
	Stress Indicators -DEH -PNBF - fibula L, R normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 527	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 532	F	30-40 yrs	168.99cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -spondylolysis - L5-bilateral -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets missing PM; lumbarization T12-both superior apophyseal facets face medial -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 539	-	1.5-2 yrs	-	
	Stress Indicators -CO - L, R normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 547	M	45+ yrs	177.95cm ± 2.99	
	Stress Indicators -CO - R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 555	M	25-34 yrs	176.39cm ± 2.99	
	Stress Indicators -DEH			

	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial -transitional vertebra thoracolumbar border - lumbar ribs L1-costal facets both side where transverse processes should be, only R rib located measuring 39.0mm long

Skeleton	Sex	Age	Stature	South Shields
CS06 559	M	40+ yrs	170.54cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L face posterior -supernumerary vertebra - S6 -pectus carinatum			

Skeleton	Sex	Age	Stature	South Shields
CS06 564	?F	30-34 yrs	159.82cm ± 3.57	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 578	F	Adult	155.41cm ± 4.45	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 579	M	45+ yrs	167.99cm ± 3.29	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L5-			

	complete

Skeleton	Sex	Age	Stature	South Shields
CS06 586	-	Birth-5 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 590	-	Birth-1 yr	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 593	-	<12 yrs	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 596	F	40-44 yrs	157.03cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6- complete but unfused at apophyseal facets -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 601	M	45+ yrs	167.03cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 606	M	45+ yrs	175.61cm ± 2.99	
	Stress Indicators -DEH -PNBF - tibia R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 613	F	Adult	158.74cm ± 4.24	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 623	?F	35-39 yrs	164.51cm ± 4.30	
	Stress Indicators -DEH			
	Congenital Defects -spondylolysis - L5-bilateral			

Skeleton	Sex	Age	Stature	South Shields
CS06 627	M	40+ yrs	149.61cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 639	F	45+ yrs	161.09cm ± 4.30	
	Stress Indicators none			
	Congenital Defects -os acromiale - L, R unobservable due to potential shoulder dislocation			

Skeleton	Sex	Age	Stature	South Shields
CS06 644	F	45+ yrs	151.47cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - lumbarization T12-L costal facet not present, R normal			

Skeleton	Sex	Age	Stature	South Shields
CS06 649	?M	20-29 yrs	177.94cm ± 4.05	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 655	-	11-12 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 666	-	26-30 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 668	-	26-30 wiu	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06	-	24-26 wiu	-	
	Stress Indicators			

673	none
	Congenital Defects none

Skeleton	Sex	Age	Stature	South Shields
CS06 678	-	~12 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects -hypoplastic lamina - C2-R, L normal, measurements: R=8.0mm, L=11.0mm, spinous process centred -hypoplastic lamina - C3-L, R normal, measurements: R=9.7mm, L=6.5mm, spinous process pushed to R side -block vertebra - T2-T3-unfused at bodies, neural arches fused, R transverse processes fused, L unfused, T2 leans inferiorly to R			

Skeleton	Sex	Age	Stature	South Shields
CS06 684	-	fetal	-	
	Stress Indicators none			
	Congenital Defects -thanatophoric dysplasia - humerus-R shortened & thickened, length=28.3mm, L missing PM -thanatophoric dysplasia - ulna-both thickened, possibly proportional in length, proximal unusually broadened, length measurements: R=27.8mm, L=27.4mm -thanatophoric dysplasia - radius-L thickened, possibly proportional in length, distal unusually broadened, length=25.9, R missing PM -thanatophoric dysplasia - femur-both shortened & thickened, distal ends higher on lateral sides, has telephone receiver appearance, length measurements: R=29.8mm, L=30.9mm -thanatophoric dysplasia - tibia-both shortened & thickened, measurements: R=26.7mm, L=25.9mm -thanatophoric dysplasia-fibula-both shortened & thickened, measurements: R=21.9mm, unable to measure L since broken PM -thanatophoric dysplasia -metacarpals or phalanges-broadened metaphyses -thanatophoric dysplasia - all metaphyses broadened -thanatophoric dysplasia - ilia-both thickened, large acetabula -thanatophoric dysplasia - ribs-ends flared, R ribs may be more compressed than L			

	-thanatophoric dysplasia - lumbar vertebrae-3 prematurely fused to body but not fused at midline, bodies flattened? -thanatophoric dysplasia - temporal-normal -thanatophoric dysplasia - mandible-normal

Skeleton	Sex	Age	Stature	South Shields
CS06 688	-	36-38 wks	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 693	-	24-28 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 701	-	32-34 wiu	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 713	-	34-38 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 718	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 751a&b	-	24-26 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 756	-	~6 months	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 766	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 774	-	32-34 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 796	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 803	-	36-38 wiu	-	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	South Shields
CS06 808	-	22-24 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 814	?M	45+ yrs	166.38cm ± 3.29	
	Stress Indicators -PNBF - tibia R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 820	-	22-24 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 825	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 832	-	26-30 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 838	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 842	-	28-32 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 847	-	36-40 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 851	-	30-32 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 857	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 862	-	18-20 wiu	-	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	South Shields
CS06 867	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 872	-	38-40 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 878	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 888	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 889	M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 898	M	Adult	178.99cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 900	M	Adult	165.61cm ± 3.27	
	Stress Indicators -PNBF - tibia R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 902	F	Adult	158.77cm ± 4.45	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-R inferior apophyseal facet faces anterior, L faces lateral; L1-missing PM			

Skeleton	Sex	Age	Stature	South Shields
CS06 906	?M	45+ yrs	173.12cm ± 3.37	
	Stress Indicators none			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	South Shields
CS06 909	F	35-39 yrs	150.68cm ± 4.24	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06	U	3 Adults	unable	

910	**Stress Indicators** -PNBF - tibia R, L unobservable -PNBF - fibula R, L normal
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-missing PM -transitional vertebra lumbosacral border - sacralization L5-complete

Skeleton	Sex	Age	Stature	South Shields
CS06 913	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 917	F	Adult	unable	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 920	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 925	F	40-44 yrs	158.98cm ± 3.55	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 926	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 928	M	25-34 yrs	176.13cm ± 3.27	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face diagonally lateral; lumbarization T12-both superior apophyseal facets face diagonally medial			

Skeleton	Sex	Age	Stature	South Shields
CS06 933	M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 935	M	Adult	175.48cm ± 4.05	
	Stress Indicators -CO - R& L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 941	F	30-34 yrs	165.65cm ± 3.55	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 945	?M	20-29 yrs	unable	
	Stress Indicators -CO - R, L normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 947	?F	45+ yrs	168.43cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -cleft neural arch - L6 -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 952	F	45+ yrs	163.84cm ± 3.55	
	Stress Indicators -CO - R, L normal			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	South Shields
CS06 954	U	Adult	unable	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula L, R unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 956	F	35-39 yrs	154.53 cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects			

	-transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L faces posterior, no costal facet present on L side, potential costal facet (rudimentary & convex) on knob on transverse process -rudimentary apophyseal facet - L5-L inferior, measurements: width R=10.8mm, L=7.0mm, length R=17.2mm, L =8.8mm -rudimentary apophyseal facet - S1-L superior, measurements: width R=11.3mm, L=5.2mm, length R=15.7mm, L=7.8mm

Skeleton	Sex	Age	Stature	South Shields
CS06 960	-	~3 yrs	-	
	Stress Indicators -CO - L, R normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 963	U	3 Adults	unable	
	Stress Indicators -PNBF - tibia L, others normal (2 R, 1 L)			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 965	?M	30-39 yrs	169.32cm ± 4.05	
	Stress Indicators -DEH			
	Congenital Defects -spondylolysis - L5-bilateral			

Skeleton	Sex	Age	Stature	South Shields
CS06 969	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 970	M	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 974	M	25-34 yrs	165.21cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete but not fused at apophyseal facets -supernumerary vertebra - L6 -absent vertebra - sacrum-only 4 segments present, within spine C4-coccyx fit together			

Skeleton	Sex	Age	Stature	South Shields
CS06 978	-	~Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 981	-	8-10 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 982	-	2-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 984	-	3-4 yrs	-	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - L5 -spondylolysis? - L2-L side normal, R side is a small triangle of superior neural arch unfused to L side, inferior lateral corner contains an articular facet facing posterior, no neural arch articular facet facing anterior & articulates with facet from triangle piece, normal inferior apophyseal facet for articulation with L3			

Skeleton	Sex	Age	Stature	South Shields
CS06 986	-	4-5 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 987	M	30-34 yrs	169.93 cm ± 4.05	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects -transitional vertebra cervicothoracic border - cervical rib C7-potential rudimentary rib, R, points inferiorly, length from body=21.1mm, height=20.0mm, L side missing PM -transitional vertebra thoracolumbar border - T12-inferior portion of neural arch missing PM; L1-both superior apophyseal facets face diagonally posterior-medial, no costal facets present, has general appearance of a T12			

Skeleton	Sex	Age	Stature	South Shields
CS06 989	M	30-34 yrs	unable	
	Stress Indicators unobservable			
	Congenital Defects none			
	Other			

	also 1 Non-Adult present

Skeleton	Sex	Age	Stature	South Shields
CS06 991	F	35-39 yrs	159.54cm ± 3.55	
	Stress Indicators -DEH -PNBF - tibia R & L -PNBF - fibula L, R normal			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6- complete but only partially fused on R apophyseal facet -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 995	U	30-39 yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 998	?F	45+ yrs	158.39cm ± 3.66	
	Stress Indicators none			
	Congenital Defects -aplastic ulnar styloid process - R & L			

Skeleton	Sex	Age	Stature	South Shields
CS06 1000	M	30-34 yrs	161.93cm ± 4.05	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1002	-	Birth-6 mos	-	
	Stress Indicators -CO - R & L			
	Congenital Defects			

	none

Skeleton	Sex	Age	Stature	South Shields
CS06 1005	?M	Adult	164.23cm ± 3.27	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1007	-	Birth-6 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1007i	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1010	M	30-34 yrs	177.04cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L faces posterior, R costal facet rudimentary at about 1/3 size of L, R rudimentary rib present, measurements: length R=54.1mm but broken PM, L=97.8mm, head height R=8.0mm, L= 13.8mm, head length R=6.8mm, L=15.2mm -absent vertebra - sacrum-only 4 segments present, T2-coccyx fit together			

Skeleton	Sex	Age	Stature	South Shields
CS06	U	Adult -		
	Stress Indicators			

1011	-PNBF - fibula R, L unobservable
	Congenital Defects none

Skeleton	Sex	Age	Stature	South Shields
CS06 1013	-	4-5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1015	?F	Adult	unable	
	Stress Indicators -CO - R, L unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1017	-	~1 yr	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1021	-	7-8 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1023a	-	8.5-10.5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 1023b	-	1-3 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 11002	?F	35-39 yrs	152.90cm ± 3.72	
	Stress Indicators none			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	South Shields
CS06 11007	?M	45+ yrs	169.63cm ± 4.05	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	South Shields
CS06 11008	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

APPENDIX D
CHURCH OF ST MICHAEL AND ST LAWRENCE, FEWSTON OSTEOLOGICAL DATA

List of Abbreviations for the Fewston Data:

CO cribra orbitalia
DEH dental enamel hypoplasia
F female
?F potential female
L left
M male
?M potential male
mos months
PH porotic hyperostosis
PM post-mortem
PNBF periosteal new bone formation
R right
U unable to determine sex
wiu weeks in utero
yrs years

Named Individual information from Caffell and Holst, 2010

Skeleton	Sex	Age	Stature	Fewston
F006	U	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F009	-	4-5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F012	?M	Adult	unable	
	Stress Indicators -DEH			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F018	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F021	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F028	-	15-17 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F031	U	Adult?	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F034	-	<1 yr	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F037	U	Adult	unable	
	Stress Indicators -CO - R, L unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F040	U	45+ yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F044	-	2-3 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F047	M	30-34 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F050	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F053	?F	45+ yrs	159.21cm ± 4.24	
	Stress Indicators -CO - R & L			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F056	-	6-8 yrs	-	
	Stress Indicators -CO - R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F059	U	33+ yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F062	-	10-11 yrs	-	
	Stress Indicators none			
	Congenital Defects -transitional vertebra cervicothoracic border - cervical rib C7-R, angled inferiorly & slightly anterior, length=14.7mm, width=11.2mm at widest, L side missing PM -transitional vertebra lumbosacral border - T12-both inferior apophyseal facets face anterior; L1-missing PM, no lumbar ribs located			

Skeleton	Sex	Age	Stature	Fewston
F065	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F068	?M	Adult	176.53cm ± 4.32	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F071	-	6-9 yrs	-	
	Stress Indicators -CO - R, L normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F074	M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F077	?M	17-25 yrs	unable	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F078	-	1-3 yrs	-	
	Stress Indicators -CO - R& L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F080	F	40-44 yrs	163.57cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F082	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F085	?M	20-29 yrs	166.82cm ± 3.37	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F088	M	35-44 yrs	170.67cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F093	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F094	-	~8 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F095	-	1.5-3 yrs	-	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F098	?F	Adult	129.68cm ± 3.72	
	Stress Indicators -PNBF - tibia R, L normal			
	Congenital Defects -transitional vertebra lumbosacral border - lumbarization S1- incomplete, PM damage, bodies appear to be separate, most of R side separate but attached at lateral end, L side damaged & missing PM, inferior apophyseal facets not fused to S2 facets			

Skeleton	Sex	Age	Stature	Fewston
F101	-	9 mos-1 yr	-	
	Stress Indicators none			
	Congenital Defects none			
	Named Individual Coffin Plate-Roman[d?] Marjerrison, 4th December 1895, 10 months (male)			

Skeleton	Sex	Age	Stature	Fewston
F104	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F107	F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F110	-	7-8 yrs	-	
	Stress Indicators			

	none
	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F113	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F116	-	~12 yrs	-	
	Stress Indicators -DEH -PNBF - fibula unsided			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F119	M	30-34 yrs	174.31cm ± 2.99	
	Stress Indicators -DEH -PNBF - fibula L, R normal			
	Congenital Defects -supernumerary vertebra - T13 -supernumerary ribs - costal facets present both sides of T13, minimum number of ribs=25			
	Named Individual Coffin Plate-Matthew Marjerrison, 25th February 1890, 38 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F121	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F122	F	45+ yrs	163.98cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -transitional vertebra occipitocervical border - precondylar facet-on anterior rim of foramen magnum, C1 missing PM, C2 normal in appearance & does not articulate with occipital condylar facets			
	Named Individual partially illegible Coffin Plate-John(?) Marjerrison, 27th February..., illegible (male?)			

Skeleton	Sex	Age	Stature	Fewston
F123	-	5-10 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F129	-	Non-Adult	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F130	M	45+ yrs	189.13cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -os acromiale - R, L normal			
	Named Individual Coffin Plate-George Lister, 19th July 1882, 66 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F132	-	10-17 yrs	-	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F135	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F138A	?F	Adult	160.63cm ± 4.24	
	Stress Indicators -CO - R & L			
	Congenital Defects none			
	Named Individual partially illegible Monument-Elizabeth Dibb(?), illegible, illegible (female)			

Skeleton	Sex	Age	Stature	Fewston
F138B	M	45+ yrs	177.33cm ± 4.05	
	Stress Indicators none			
	Congenital Defects -hypoplastic lamina - L6-L -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6 -elongated styloid process - R=20.8mm broken PM, L=25.8mm broken PM, both have smooth contour & angle anteriorly & slightly medially			
	Named Individual partially illegible Monument-James Dibb (?), illegible, illegible (male)			

Skeleton	Sex	Age	Stature	Fewston
F141	U	Adult	unable	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F144	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F147	F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F150	?M	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			
	Named Individual Monument-Joseph Patterson, 10th March 1860, 82 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F153	?M	45+ yrs	179.46cm ± 3.27	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F156	?F	Adult	168.85cm ± 4.45	
	Stress Indicators -DEH			
	Congenital Defects none			

	Named Individual Monument-Christiana Patterson, 23rd October 1854, 66 yrs (female)

Skeleton	Sex	Age	Stature	Fewston
F159	?F	45+ yrs	unable	
	Stress Indicators none			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	Fewston
F162	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F165	?M	45+ yrs	unable	
	Stress Indicators -DEH -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects -asymmetrical cranium - facial region turned towards R, foramen magnum skewed to R, coronal suture possibly located too far posteriorly, as move posteriorly occipital sulcus & internal occipital crest go R, as move anteriorly sagittal sulcus & frontal crest go L, some changes may be due to PM compression -asymmetrical mandible - L ramus noticeably shorter than R, measurements: inferior edge to top of condyle R=50.3mm, L=46.9mm, inferior edge to top of coronoid process R=57.3mm, L=59.1mm, L mandibular condyle smaller than R, measurements lateral to medial: R=17.8mm, L=14.2mm, superior surface of L mandibular condyle porotic, large porotic area on L mandibular fossa, R ramus & coronoid process angle laterally, L goes almost straight up -os acromiale - R, L unobservable			

Skeleton	Sex	Age	Stature	Fewston
F168	?F	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F171	?F	Adult	unable	
	Stress Indicators -PNBF - fibula R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F174	?F	30-34 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F177	F	25-39 yrs	166.62cm ± 3.55	
	Stress Indicators -PNBF - tibia R & L			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F180	M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F186	M	45+ yrs	173.03cm ± 3.27	
	Stress Indicators none			

	Congenital Defects -craniosynostosis - R side of lambdoidal suture not present, coronal suture runs straight across from L to sagittal suture than runs diagonally posterior, R side of frontal projects more anteriorly than L, L side occipital projects more posteriorly than R -transitional vertebra lumbosacral border - L6-complete -supernumerary vertebra - L6 -facet tropism - L6-L superior faces posterior, R superior faces medial -rotated apophyseal facet - L5-L inferior faces mostly anterior, R missing PM

Skeleton	Sex	Age	Stature	Fewston
F188	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F192	?M	17-29 yrs	179.42cm ± 3.37	
	Stress Indicators -CO - L, R normal -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F195	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F198	U	Adult	unable	
	Stress Indicators -PNBF - tibia R & L			
	Congenital Defects none			

	Named Individual Monument-Hannah Holmes?, -, 29 yrs (female)

Skeleton	Sex	Age	Stature	Fewston
F204	-	<11 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F205	-	~3 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F206	-	30-34 wiu	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F208	-	11-12 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F211	U	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F214	F	17-25 yrs	unable	
	Stress Indicators -CO - L, R unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F217	-	~15 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F220	?M	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F223	-	12-15 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F226	M	45+ yrs	183.93cm ± 2.99	
	Stress Indicators -PNBF - tibia R, L normal -PNBF - fibula R, L normal			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete?, PM damage to body so uncertain is completely fused there -supernumerary vertebra - L6			

	Named Individual Coffin Plate-David Lister, 16th April 1888, 84 yrs (male)

Skeleton	Sex	Age	Stature	Fewston
F229	-	~12 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F232	-	5-10 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F235	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			
	Named Individual Monument-Thomas Patterson?, 31st January 1865?, 70 yrs? (male?)			

Skeleton	Sex	Age	Stature	Fewston
F238	?F	30-34 yrs	unable	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-L inferior apophyseal facet faces mostly anterior, R faces diagonally lateral; lumbar rib? L1-L superior apophyseal facets faces diagonally posterior, R faces medial, no costal facets present, L transverse process unusually long (41.1mm)=lumbar rib?, R side missing PM -pectus carinatum			
	Named Individual			

	Coffin Plate-[Eliza?]beth [D?]emaine, ? April 1888, 49 yrs (female?)

Skeleton	Sex	Age	Stature	Fewston
F241	M	Adult	178.92cm ± 3.37	
	Stress Indicators none			
	Congenital Defects -Klippel-Feil syndrome - C3-C4			

Skeleton	Sex	Age	Stature	Fewston
F244	-	~18 mos	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F247	-	<13 yrs	-	
	Stress Indicators -PNBF - fibula R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F250	-	~12 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F253	?M	30-34 yrs	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F259	U	Adult	unable	

	Stress Indicators none
	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F262	-	11-15 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F265	?F	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F268A	-	9-10 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F271	F	17-20 yrs	unable	
	Stress Indicators -CO - R & L -DEH -PNBF - tibia R, L unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F274	U	Adult	unable	
	Stress Indicators			

	unobservable
	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F277	?F	17-25 yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F279	-	2-3 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F283	F	18-25 yrs	unable	
	Stress Indicators -CO - R, L normal -DEH			
	Congenital Defects none			
	Named Individual Monument (very tentative)-Mary Ann Lister?, 15th February 1877?, 20 yrs? (female?)			

Skeleton	Sex	Age	Stature	Fewston
F289	?F	35-39 yrs	unable	
	Stress Indicators none			
	Congenital Defects -scoliosis? - 2 unknown thoracic bodies-unequal thickness of pedicles, 1st R pedicle thinner & lower half of body seems to lean to R, 2nd L pedicle thinner & lower half of body seems to lean to L, thoracic & lumbar regions very poorly preserved & fragmentary			

Skeleton	Sex	Age	Stature	Fewston
F292	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F295	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F297	-	Non-Adult	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F300	F	45+ yrs	162.32cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -aplastic lamina - S1-L, R normal, resulted in normal cleft neural arch			
	Named Individual Monument-Mary Darnbrook?, 7th September 1870?, 78 yrs? (female?)			

Skeleton	Sex	Age	Stature	Fewston
F303	?F	20-24 yrs	157.43cm ± 4.45	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F306	F	35-39 yrs	163.81cm ± 4.45	
	Stress Indicators -PNBF - tibia L, R unobservable			
	Congenital Defects -os acromiale - L, R normal			

Skeleton	Sex	Age	Stature	Fewston
F307	M	Adult	166.90cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-both inferior apophyseal facets face diagonally anterior-lateral; L1-R superior apophyseal facet faces diagonally posterior-medial, L missing PM, no costal facets present			
	Named Individual Monument-Joseph Darnbrook?, 7th March 1869?, 78 yrs? (male?)			

Skeleton	Sex	Age	Stature	Fewston
F310	F	45+ yrs	154.67cm ± 3.55	
	Stress Indicators none			
	Congenital Defects none			
	Named Individual Coffin Plate-Mary Dickinson, 6th March 1888, 66 yrs (female)			

Skeleton	Sex	Age	Stature	Fewston
F316	?M	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F319	?F	17-25 yrs	unable	
	Stress Indicators -DEH			

	Congenital Defects none
	Named Individual Monument-Sarah Darnbrook, 26th May 1854, 23 yrs (or 22?) (female)

Skeleton	Sex	Age	Stature	Fewston
F322	-	7-8 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F325	F	45+ yrs	unable	
	Stress Indicators -CO - R, L unobservable			
	Congenital Defects none			
	Named Individual Coffin Plate-Grace Hutton, 3rd April 1921, 73 yrs (female)			

Skeleton	Sex	Age	Stature	Fewston
F328	?F	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F331	-	7-15 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F334	-	10-12 yrs	-	
	Stress Indicators			

	-CO - R & L -DEH
	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F335	-	5-11 yrs	-	
	Stress Indicators -CO - L, R unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F338	-	~15 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F339	M	35-44 yrs	171.45cm ± 2.99	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-incomplete L -supernumerary vertebra - L6			
	Named Individual Coffin Plate-Richard Gill, 11th February 1884, 41 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F342	U	Adult	unable	
	Stress Indicators -CO - R, L unobservable -DEH			
	Congenital Defects none			
	Named Individual			

	Coffin Plate-Bentley Darnbrook, 1st November 1862, 26 yrs (male)

Skeleton	Sex	Age	Stature	Fewston
F345	-	14-18 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects -hypoplastic lamina - C1-L, R normal, centre of neural arch pulled to L			

Skeleton	Sex	Age	Stature	Fewston
F348	?F	17-25 yrs	153.75cm ± 3.66	
	Stress Indicators -CO - R, L normal -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-R inferior apophyseal facet faces anterior, L damaged PM but appears to face anterior; L1-R superior apophyseal facet faces diagonally posterior-medial, L missing PM, no costal facet on R side, L side damaged PM -transitional vertebra lumbosacral border - lumbarization S1-complete			

Skeleton	Sex	Age	Stature	Fewston
F351	M	45+ yrs	174.70cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -craniosynostosis - absent squamosal suture on R & occipito-mastoid suture on L, slight asymmetry of cranium, L parietal bosses out a little further than R -transitional vertebra lumbosacral border - lumbarization S1-incomplete R, PM damaged makes observation difficult			
	Named Individual Monument-John Dickinson?, 18th August 1875 (?), 63 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F353	-	Non-Adult	-	
	Stress Indicators unobservable			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F354	-	8-9 yrs	-	
	Stress Indicators -CO - L, R unobservable -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F357	-	1-2 yrs	-	
	Stress Indicators -PH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F360	M	45+ yrs	177.56cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -bridged rib - unknown side or location, length=11.4mm, width=11.3mm, external surface of bridge smooth, interior is lumpy, rib attached via bridge broken off PM -os acromiale - R, L unobservable			
	Named Individual Coffin Plate-Gill Wigglesworth, 24th April 1886, 67 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F363	F	45+ yrs	165.15cm ± 4.45	
	Stress Indicators -PNBF - tibia L, R normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-R inferior apophyseal facet missing PM, L faces lateral; lumbar ribs L1-R superior apophyseal facet missing PM, L faces medial, costal facets R & L, ribs present both sides -transitional vertebra lumbosacral border - sacralization L6-			

	complete -supernumerary vertebra - L6
	Named Individual Coffin Plate-Sarah Gill, 13th November 1889, 54 yrs (female)

Skeleton	Sex	Age	Stature	Fewston
F366	M	40+ yrs	174.57cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - C1 -cleft neural arch - L6 -spondylolysis - L5-bilateral -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			
	Named Individual Coffin Plate-John Renton Newsome, 3rd February 1892, 76 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F369	?F	Adult	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F372	?F	45+ yrs	unable	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	Fewston
F375	-	5-6 yrs	-	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F378	F	30-34 yrs	151.89cm ± 3.55	
	Stress Indicators none			
	Congenital Defects none			
	Named Individual Coffin Plate-Elina Wigglesworth, 27th February 1895, 34 yrs (female)			

Skeleton	Sex	Age	Stature	Fewston
F384	?F	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F387	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F396	?F	Adult	unable	
	Stress Indicators -CO - R, L unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F399	U	Adult?	unable	
	Stress Indicators -PNBF - tibia L, R unobservable			
	Congenital Defects			

	none

Skeleton	Sex	Age	Stature	Fewston
F402	U	Adult?	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F405	U	Unknown	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F408	M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			
	Named Individual Coffin Plate-Richard Gill, 18th May 1883, 78 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F414	?M	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F417	-	2-5 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F420	-	2-4 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F423	-	12-15 yrs	-	
	Stress Indicators -DEH -PNBF - fibula R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F426	?F	25-29 yrs	163.29cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet angled to face mostly lateral, R faces anterior; lumbarization T12-L superior apophyseal facet angled to face mostly medial, R faces posterior, both costal facets small, transverse processes short & posterior			
	Named Individual Monument (unlikely ID)-Joseph Wilson?, 4th January 1871?, 25 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F429	-	1.5-2 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F432	U	Adult	unable	
	Stress Indicators none			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Fewston
F435	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F438	-	15-18 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F441	?M	Adult	unable	
	Stress Indicators -CO - R, L normal			
	Congenital Defects none			
	Named Individual Monument-Daniel Fox, 19th March 1884?, 33 yrs (male)			

Skeleton	Sex	Age	Stature	Fewston
F444	-	>4 <18 yrs	-	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F447	?F	17-25 yrs	unable	
	Stress Indicators -CO - R, L unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F450	?F	45+ yrs	unable	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-L superior apophyseal facet faces medial, R faces posterior			

Skeleton	Sex	Age	Stature	Fewston
F453	U	Adult	unable	
	Stress Indicators unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Fewston
F456	?M	45+ yrs	unable	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet missing PM, L faces posterior			

Skeleton	Sex	Age	Stature	Fewston
F459	M	40+ yrs	184.58cm ± 2.99	
	Stress Indicators -PNBF - tibia R, L normal -PNBF - fibula R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T12-both inferior apophyseal facets face lateral; lumbarization T13-both superior apophyseal facets face medial, costal facets both sides -supernumerary vertebra - T13 -supernumerary ribs - T13 has costal facets both sides, no additional ribs found			

Skeleton	Sex	Age	Stature	Fewston
F460	F	Adult	unable	

	Stress Indicators none
	Congenital Defects none

APPENDIX E
ST MARTIN'S CHURCH, WHARRAM PERCY
OSTEOLOGICAL DATA

List of Abbreviations for the Wharram Percy Data:

CO cribra orbitalia
DEH dental enamel hypoplasia
F female
?F potential female
L left
M male
?M potential male
MNI minimum number of individuals
PH porotic hyperostosis
PM post-mortem
PNBF periosteal new bone formation
R right
U unable to determine sex
wiu weeks in utero
yrs years

Radiocarbon Date information from Mays, 2007b

Skeleton	Sex	Age	Stature	Wharram Percy
CN01	M	Adult	165.93cm ± 4.05	
	Stress Indicators -PH			
	Congenital Defects -os acromiale - L, R unobservable			

Skeleton	Sex	Age	Stature	Wharram Percy
CN02	?M	Adult	162.22cm ± 2.99	
	Stress Indicators -CO - R & L			
	Congenital Defects -os acromiale - R, L unobservable -aplastic ulnar styloid process - R, L missing PM			

Skeleton	Sex	Age	Stature	Wharram Percy
CN03	F	Adult	unable	
	Stress Indicators unobservable			

	Congenital Defects none
	Radiocarbon Date 1420-1640 AD

Skeleton	Sex	Age	Stature	Wharram Percy
CN04	-	6-7 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN05	M	45+ yrs	171.45cm ± 2.99	
	Stress Indicators -PNBF - fibula R, L normal			
	Congenital Defects -cleft neural arch - C1 -transitional vertebra thoracolumbar border - T12-R inferior apophyseal facet faces anterior, L faces lateral; L1-R superior apophyseal facet faces posterior, L faces medial, no costal facets present -transitional vertebra lumbosacral border - sacralization L5-partial R			

Skeleton	Sex	Age	Stature	Wharram Percy
CN06	M	25-29 yrs	172.56cm ± 3.27	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN07	?F	40-49 yrs	165.37cm ± 3.55	
	Stress Indicators -CO - R, L normal -PNBF - tibia R			
	Congenital Defects -cleft neural arch - S3 -rudimentary costal facet - T12 L			

	-rudimentary rib - possibly R

Skeleton	Sex	Age	Stature	Wharram Percy
CN09	-	4-11 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN11	M	20-24 yrs	173.66cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN12	M	30-34 yrs	167.55cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-complete -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	Wharram Percy
CN13	F	30-39 yrs	165.65cm ± 3.55	
	Stress Indicators -CO - R & L			
	Congenital Defects -supernumerary vertebra - S6			

Skeleton	Sex	Age	Stature	Wharram Percy
CN14	M	30+ yrs	170.15cm ± 2.99	
	Stress Indicators -PNBF - fibula R & L			
	Congenital Defects -transitional vertebra thoracolumbar border - lumbarization T12-R & L superior apophyseal facets face medial, costal and inferior apophyseal facets normal			

Skeleton	Sex	Age	Stature	Wharram Percy
CN15	-	~12 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN16	F	40+ yrs	159.54cm ± 3.55	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN17	M	30-34 yrs	174.83cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN18	M	45+ yrs	174.31cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial			

Skeleton	Sex	Age	Stature	Wharram Percy
CN19	F	45+ yrs	168.22cm ± 4.24	
	Stress Indicators -CO - R, L normal			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-L superior apophyseal facet faces posterior, R faces medial			

Skeleton	Sex	Age	Stature	Wharram Percy
CN20	?F	30-34 yrs	160.37cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-incomplete R -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	Wharram Percy
CN22	?F	Adult	156.36cm ± 3.72	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN24	M	45+ yrs	171.06cm ± 2.99	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial -transitional vertebra lumbosacral border - sacralization L5-complete			

Skeleton	Sex	Age	Stature	Wharram Percy
CN27	M	40-44 yrs	166.64cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN28	M	25-34 yrs	166.12cm ± 2.99	
	Stress Indicators -DEH -PNBF - tibia R & L -PNBF - fibula L, R normal			

	Congenital Defects none

Skeleton	Sex	Age	Stature	Wharram Percy
CN29	F	40-44 yrs	153.15cm ± 3.72	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN30	?F	20-24 yrs	161.48cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN31	?M	40-44 yrs	169.59cm ± 3.37	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN32	M	35-39 yrs	170.89cm ± 3.27	
	Stress Indicators -DEH -PNBF - fibula R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN33	M	45+ yrs	173.14cm ± 2.99	
	Stress Indicators -PNBF - tibia R & L -PNBF - fibula R & L			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-			

	L superior apophyseal facet faces medial, R faces posterior

Skeleton	Sex	Age	Stature	Wharram Percy
CN36	?F	40-44 yrs	159.08cm ± 3.72	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN37	M	40-44 yrs	172.36cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-L inferior apophyseal facet faces anterior, R missing PM; lumbarization T12-R superior apophyseal facet faces medial, L missing PM			

Skeleton	Sex	Age	Stature	Wharram Percy
CN38	M	30-34 yrs	170.93cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN39	M	40-44 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
CN40	?F	45+ yrs	165.37cm ± 3.55	
	Stress Indicators -CO - R & L			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization lowest lumbar vertebra-partial R (no lumbar vertebra present, diagnosed based on changes to sacrum)			

Skeleton	Sex	Age	Stature	Wharram Percy
CN41	F	40-44 yrs	163.84cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -transitional vertebra thoracolumbar vertebra - T11-L inferior apophyseal facet faces lateral, R faces anterior; lumbarization T12-L superior apophyseal facet faces medial, R missing PM -transitional vertebra lumbosacral vertebra - sacralization L5-partial L -supernumerary vertebra - S6			

Skeleton	Sex	Age	Stature	Wharram Percy
CN42	?F	45+ yrs	152.17cm ± 3.55	
	Stress Indicators -PNBF - fibula L, R missing PM			
	Congenital Defects -os acromiale - R, L normal			

Skeleton	Sex	Age	Stature	Wharram Percy
CN43	M	30-34 yrs	165.31cm ± 3.37	
	Stress Indicators none			
	Congenital Defects -cleft neural arch - S1, S3-S5 -transitional vertebra thoracolumbar border - T11-missing PM; lumbarization T12-both superior apophyseal facets face medial -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	Wharram Percy
CN44	M	40-44 yrs	167.16cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			
	Radiocarbon Date 1640-1955 AD			

Skeleton	Sex	Age	Stature	Wharram Percy
CN45	M	40-44 yrs	170.89cm ± 3.27	

	Stress Indicators unobservable
	Congenital Defects -rib spur - possibly R, 14mm from sternal end, 5mm long

Skeleton	Sex	Age	Stature	Wharram Percy
CN46	U	unable	unable	
	Stress Indicators -DEH			
	Congenital Defects none			
	Other MNI=2 adults			

Skeleton	Sex	Age	Stature	Wharram Percy
CN47	-	1-3 yrs	-	
	Stress Indicators -CO - L, R unobservable			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
SA02	M	25-34 yrs	162.63cm ± 3.29	
	Stress Indicators -DEH -PNBF - tibia R & L			
	Congenital Defects none			
	Radiocarbon Date 1440-1660 AD			

Skeleton	Sex	Age	Stature	Wharram Percy
SA03	M	45+ yrs	174.70cm ± 3.27	
	Stress Indicators -DEH			
	Congenital Defects none			
	Radiocarbon Date 1430-1660 AD			

Skeleton	Sex	Age	Stature	Wharram Percy
SA17	F	25-34 yrs	160.51cm ± 3.55	
	Stress Indicators -DEH			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
SA23	F	45+ yrs	150.50cm ± 3.55	
	Stress Indicators none			
	Congenital Defects -block vertebra - T4-T5 -os acromiale - L, R unobservable			

Skeleton	Sex	Age	Stature	Wharram Percy
SA33	?M	40-44 yrs	162.22cm ± 2.99	
	Stress Indicators none			
	Congenital Defects none			
	Radiocarbon Date 1420-1650 AD			

Skeleton	Sex	Age	Stature	Wharram Percy
SA45	-	12-16 yrs	-	
	Stress Indicators -CO - R & L -DEH			
	Congenital Defects -supernumerary vertebra - L6 -absent vertebra - only six cervical vertebrae			

Skeleton	Sex	Age	Stature	Wharram Percy
SA48	F	30-34 yrs	161.58cm ± 3.66	
	Stress Indicators -CO - R & L			
	Congenital Defects -transitional vertebra thoracolumbar border - T11-missing PM;			

	lumbarization T12-R superior apophyseal facet faces medial, L faces posterior, L inferior apophyseal facet faces lateral, R faces anterior -transitional vertebra thoracolumbar border - lumbar ribs L1-L superior apophyseal facet faces medial, R faces posterior, bilateral costal facets in place of transverse processes, no lumbar ribs recovered

Skeleton	Sex	Age	Stature	Wharram Percy
SA49	F	45+ yrs	159.84cm ± 3.66	
	Stress Indicators -PNBF - tibia R, L normal			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
V01	-	36-38 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
V03	-	36-38 wiu	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
V06	-	Birth	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
V07	-	12-15 yrs	-	
	Stress Indicators -DEH			
	Congenital Defects			

	none

Skeleton	Sex	Age	Stature	Wharram Percy
V16	U	Adult	unable	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra lumbosacral border - sacralization L6-partial L, R unobservable -supernumerary vertebra - L6			

Skeleton	Sex	Age	Stature	Wharram Percy
V20	-	1.5-2 yrs	-	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
V21	U	Adult	unable	
	Stress Indicators none			
	Congenital Defects none			

Skeleton	Sex	Age	Stature	Wharram Percy
V42	M	40-44 yrs	175.35cm ± 2.99	
	Stress Indicators -DEH			
	Congenital Defects -transitional vertebra cervicothoracic border - cervical rib C7-R, L normal, 26.8mm long but end broken off PM -transitional vertebra thoracolumbar border - lumbar ribs L1-bilateral costal facets located on transverse processes -rudimentary transverse process - T1-bilateral			

Skeleton	Sex	Age	Stature	Wharram Percy
WCO010	?M	18-23 yrs	unable	
	Stress Indicators -DEH			

	Congenital Defects -transitional vertebra cervicothoracic border - cervical rib C7-R, L side missing PM, rib facet on transverse process, transverse foramen absent R and L -transitional vertebra thoracolumbar border - T11-R inferior apophyseal facet faces lateral, L faces anterior; lumbarization T12-R superior apophyseal facet faces medial, L faces posterior, rudimentary L costal facet, rudimentary rib
	Radiocarbon Date 1420-1640 AD

Skeleton	Sex	Age	Stature	Wharram Percy
WCO044	-	7.5-12 yrs	-	
	Stress Indicators none			
	Congenital Defects none			
	Radiocarbon Date 1440-1800 AD			

Skeleton	Sex	Age	Stature	Wharram Percy
WCO045	F	45+ yrs	162.74cm ± 3.66	
	Stress Indicators none			
	Congenital Defects -supernumerary vertebra - L6			
	Radiocarbon Date 1520-1920 AD			

Skeleton	Sex	Age	Stature	Wharram Percy
WCO153	U	35-39 yrs	unable	
	Stress Indicators none			
	Congenital Defects none			
	Radiocarbon Date 1430-1660 AD			

Skeleton	Sex	Age	Stature	Wharram Percy
WCO170	U	45+ yrs	unable	

	Stress Indicators -CO - R, L normal
	Congenital Defects -transitional vertebra thoracolumbar border - T11-both inferior apophyseal facets face lateral; lumbarization T12-both superior apophyseal facets face medial, possible rudimentary rib fused to L body, R costal facet on transverse process
	Radiocarbon Date 1330-1640 AD

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